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A REPORT ON THE VALUE OF THE IVY BLEEDING TIME TEST AND THE USE OF VIOSTEROL IN CASES OF OBSTRUCTIVE JAUNDICE

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THAT the problem of the bleeding tendency in cases of obstructive jaundice is a serious matter is appreciated when one realizes that some 50 per cent of all postoperative deaths among these patients is said to be the result of hemorrhage. Many tests have been recommended in the past for the determination of the latent bleeding tendency in jaundiced patients, but no reliable and practical one from a clinical point of view was discovered until recently. In April, 1935, Ivy[1] and McNealy and their coworkers announced a new bleeding time test which seemed to fulfill these requirements. In a word, the test was a modification of the old Duke test with the factor of capillary wall tonicity eliminated by means of a blood pressure cuff. Finding clinical evidence to support the hypothesis that the cause of bleeding in obstructive jaundice is due to a low liver reserve of fat soluble vitamins, McNealy[2] and others advocated at this time the use of viosterol for the correction or prevention of the bleeding tendency in this type of jaundice. Their clinical results were based upon observations made on 900 miscellaneous cases followed at the Cook County Hospital in Chicago and were excellent indeed. We proposed to try out both the test (now called the Ivy bleeding time test) and the use of viosterol in the cases of obstructive jaundice seen on the surgical services at the University Hospital and see whether or not their clinical results could be substantiated.

During the past year a series of 35 cases of obstructive jaundice, with and without bleeding, has been carefully followed and studied by us. Each of these patients was tested by the same individual and all clinical data were obtained and recorded by him. Specific and identical notes were kept for all cases.

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The following chart gives the sex, age distribution, and diagnoses of the cases studied:

SEX	TOTAL	AGE YOUNGEST	AGE OLDEST	AGE AVERAGE	CD OR GB STONES	PANCREATIC, OR C.D. BILIARY CARCINOMA	STRICTURE, OR STENOSIS	CIRRHOSIS
Male	14	25	70	55	6 (43%)	7 (50%)	–	1 (7%)
Female	21	25	71	51	10 (40%)	9 (44%)	2 (8%)	–

I

We were first interested in evaluating the coagulation and bleeding time factors. The normal coagulation time was considered under 5 minutes. The upper limit of normal for the Duke bleeding time was considered to be 180 seconds and that for the Ivy test 240 seconds, as determined by Ivy and McNealy. Our observations in this regard were as follows:

	CASES	ELEVATED COAG. TIME	ELEVATED DUKE B.T.	ELEVATED IVY B.T.
With bleeding	9	0	4 (44%)	9 (100%)
Without bleeding	26	0	5 (20%)	15 (60%)

In no case, it will be observed, was the coagulating time significantly elevated. However, bleeding time elevation, especially the Ivy, signified potential bleeding. In the cases with bleeding, the Duke bleeding time was elevated in 44 per cent and the Ivy in 100 per cent. In cases not presenting clinical bleeding, the Duke bleeding time was elevated in 20 per cent while the Ivy was elevated in 60 per cent. Comparing the two bleeding time tests, it is seen that the Ivy test had a prognostic value not observed with the Duke test.

II

Having found to our own satisfaction a definite correlation between the bleeding time and clinical bleeding, we next made observations regarding the Ivy bleeding time and the jaundice itself.

	CASES	ELEVATED IVY B.T.	NORMAL IVY B.T.
With bleeding	9	9 (100%)	0
Without bleeding	21	12 (57%)	9 (43%)

This chart showed that in these cases of obstructive jaundice, with or without bleeding, the Ivy bleeding time was usually elevated.

The figures just mentioned suggested that perhaps there existed a definite relationship between the elevated Ivy bleeding time and the degree or duration of the jaundice. In a total of 30 of the cases it was possible to determine accurately the total number of weeks' duration of the jaundice in the past and present illnesses of these patients.

Jaundiced Cases With Bleeding

	CASE	IVY	BLEEDING PRESENT	BLEEDING LATER*	JAUNDICE DEGREE†	JAUNDICE DURATION	BILIRUBIN‡
1.	GB	350	x	x	3	2 wk.	100
2.	MB	335	o	x	3	9 wk.	180
3.	JE	540	o	x	3	6 mo.	108
4.	GG	330	o	x	2	3 mo.	90
5.	EP	360	o	x	2	7 mo.	60
6.	MK	480	x	x	1	30 mo.	30
7.	IW	330	o	x	3	4 wk.	160
8.	CL	245	o	x	3	5 mo.	160
9.	HA	375	x	x	3	2 wk.	200

Jaundiced Cases Without Bleeding

	CASE	IVY	BLEEDING PRESENT	BLEEDING LATER*	JAUNDICE DEGREE†	JAUNDICE DURATION	BILIRUBIN‡
10.	KA	450	o	o	3	2 mo.	100
11.	CT	700	o	o	2	1 wk.	55
12.	HO	360	o	o	1	4 wk.	–
13.	WE	360	o	o	1	3 mo.	15
14.	KC	460	o	o	3	5 mo.	176
15.	ES	430	o	o	1	2 days	20
16.	JK	250	o	o	1	4 mo.	70
17.	MH	195	o	o	1	4 wk.	18
18.	McM	310	o	o	1	5 wk.	50
19.	RR	200	o	o	2	6 wk.	100
20.	NC	105	o	o	2	5 mo.	80
21.	EF	175	o	o	3	2 wk.	100
22.	JD	355	o	o	3	3 mo.	220
23.	SC	400	o	o	3	2 mo.	110
24.	JW	250	o	o	3	3 wk.	120
25.	VG	200	o	o	1	4 wk.	13
26.	LM	120	o	o	2	12 mo.	18
27.	FC	210	o	x§	2	10 wk.	50
28.	EB	170	o	o	2	2 wk.	90
29.	IE	240	o	o	1	6 wk.	30
30.	FV	300	o	o	1	3 wk.	25

*Developing after present examination.
†This is the clinician's estimation; 1 is mild; 2 is moderate; 3 is marked.
‡All of the bilirubin tests were "direct."
§This patient developed p.o. mesenteric thrombosis with gangrene of ileum.

Cases with a lower jaundice (and bilirubin) but with a longer duration, as in Case 5, had about the same bleeding time as those with a higher jaundice and a shorter duration, as in Case 1. Granting the hypothesis of Ivy and McNealy as to the cause of bleeding in these jaundice cases, we believed that these figures indicated that the rate of depletion of the liver reserve of fat soluble vitamins varies with, and depends upon, the duration of the obstruction and upon its completeness.

EFFECT ON THE B.T. AND BLEEDING BY THE SURGICAL REMOVAL OF THE OBSTRUCTION CAUSING THE JAUNDICE WITHOUT THE USE OF VIOSTEROL

	NAME	DAYS P.O.	IVY B.T.	BLEEDING	BILIRUBIN
1.	MB	6	480 to 260	0 to 0	181 to 65
2.	FV	9	300 to 160	0 to 0	25 to 12
3.	ES	5	430 to 150	0 to 0	20 to –
4.	IE	5	260 to 160	0 to 0	30 to 15

In 8 cases, where viosterol was not used, observations were made regarding the bleeding time and the surgical removal, or failure of removal, of the cause of the obstruction.

THE SAME EFFECTS WHEN SURGICAL REMOVAL OF THE OBSTRUCTION COULD NOT BE DONE

1.	LW	10	290 to 480	0 to x	160 to 160
2.	CL	5	245 to 360	0 to x	150 to 160
3.	GG	15	330 to –	0 to x	80 to 60
4.	NC	6	110 to 210	0 to 0	80 to 50

The surgical removal alone of the obstruction caused a progressive and rapid diminution in the bleeding time and the bilirubin. Where only a laparotomy was performed and the obstruction could not be removed (cases with extensive and progressive carcinomatosis or hepatitis), the Ivy bleeding time increased and the bilirubin did not diminish. We considered this to be more evidence that the Ivy bleeding time is related to the obstructive element in the jaundice.

III

It was possible to follow carefully 20 cases in which viosterol was used, with or without bile salts, as recommended by Ivy and McNealy. Each of these patients was given 50 minims of viosterol (Mead, Johnson & Company) three times a day and 5 or 10 gr. tablet of ox or pig bile by mouth three times a day when the stools were acholic.

		BEFORE VIOSTEROL					VIOSTEROL	AFTER VIOSTEROL				
	CASE	J.*	BIL.*	BL.*	DUKE	IVY	50 M. T.I.D.	J.	BIL.	BL.	DUKE	IVY
1.	GB	3	100	x	600	600	3 days	4	120	xx	280	620‡
2.	KA	3	100	o	180	450	6 days†	4	180	o	210	540‡
3.	MB	3	180	o	120	335	6 days	3	–	o	150	200
4.	CT	2	55	o	210	700	5 days	1	7	o	130	280
5.	JE	3	108	o	300	540	11 days	2	60	o	220	340
6.	GG	3	90	o	170	330	6 days	3	80	o	180	350‡
7.	FV	o	20	o	–	300	8 days	o	–	o	80	100
8.	HO	1	35	o	160	360	10 days†	1	12	o	115	160
9.	KC	3	–	o	180	460	10 days	3	176	o	160	240
10.	PK	o	o	o	130	350	5 days	o	1	o	180	200
11.	WE	1	15	o	120	360	5 days	1	–	o	130	160
12.	EP	2	60	o	360	420	7 days	3	150	x	300	360
							26 days†	3	180	x	–	480
							37 days†	2	–	o	–	333
							41 days†	1	70	o	–	270
13.	ES	1	20	o	225	450	14 days*	1	–	o	60	110
14.	JKN	2	70	o	200	280	3 days*	1	8	o	70	160
15.	MK	2	30	x	300	480	20 days	2	34	less	360	420
							48 days*	2	45	less	–	190
16.	MH	1	18	o	60	195	7 days	less	–	o	55	180
17.	McM	1	–	o	60	310	4 days	1	50	o	160	195
							20 days	less	–	o	55	180
18.	IW	3	160	o	–	330	11 days*	4	225	o	240	300
19.	NM	o	–	o	90	270	10 days	o	–	o	60	210
20.	EF	3	100	o	55	175	10 days	3	–	o	180	190

*J.—jaundice; Bil.—bilirubin, Bl.—bleeding.

†This means with bile salts.

‡These were cases of carcinoma with increase in size of lesion.

It is to be observed from this chart that the use of viosterol in almost all of our cases of obstructive jaundice definitely diminished the Ivy bleeding time factor and the incidence of bleeding. In 13 cases the Ivy time was reduced to normal. In 4 cases the reduction was toward normal. In 1 case there was no change. In 2 cases there was an increase, and in both instances these patients had extensive carcinomatosis.

Rearrangement and correlation of the cases and facts just reviewed with deaths indicated that the use of viosterol has a certain prognostic value.

	IVY B.T. REDUCTION		DEATHS	DAYS VIOSTEROL	AVERAGE DAYS USED
With bleeding	To normal	0	0		
(8 cases)	Toward normal	4	3	6-17	11
	No change	0	0		
	Increased	4	4		
Without bleeding	To normal	7	0		
(12 cases)	Toward normal	4	0	3-20	10
	No change	1	0		
	Increased	0	0		

Therefore, in cases of obstructive jaundice without clinical bleeding, the prognosis was considered good when reduction of the Ivy bleeding time to or toward normal occurred after an adequate trial of viosterol. Only rarely was there an increase in the bleeding time among these patients. In obstructive jaundice cases with clinical bleeding and presenting a reduction of the Ivy bleeding time to normal with the use of viosterol, the prognosis was likewise fairly good. But, the prognosis in such cases was only fair when the bleeding time returned merely toward normal, poor when it showed no change, and fatal when the Ivy time increased. These bleeding time changes occurred, one way or the other, within a period of 7 to 21 days, the average time being about 10 days.

IV

Fifteen of the 35 cases died during this past year, either in or outside the hospital. In 12 of them an autopsy was obtained with the microscopic liver findings shown in the table on page 822.

It will be noted that the chief autopsy diagnoses were quite evenly divided between carcinoma and cirrhosis of the liver. May it not be argued, as a diagnostic point, that when the Ivy bleeding time does not improve in a reasonable period of time with the use of viosterol, carcinoma or cirrhosis of the liver is to be suspected as the likely cause of the supposedly permanent liver fat soluble vitamin insufficiency? Again, may it not be similarly said postoperatively that the failure to show an improved Ivy bleeding time after apparent removal of the obstruction causing the jaundice (as best as can be established by palpation and

	CASE	HEMORR.	CA.	CIRR.	CHOL.	INTRA-H.	PERI-H.	ATROPHY	FATTY	OBST. J.
					*Clinical Cases With Bleeding**					
1.	CL	x	(X)	o	x	x	o	o	o	x
2.	IW	x	o	(X)	x	x	o	o	o	x
3.	EP	x	o	o	(X)†	o	o	o	o	x
4.	GB	x	(X)	o	o	o	o	x	x	x
5.	HA	x	o	(X)	o	o	o	o	o	x
6.	MK	x	o	(X)	x	o	x	o	o	x
7.	JE	x	(X)	(X)	o	o	o	o	x	x
					(7 out of 9 deaths, all with autopsy)					
					Clinical Cases Without Bleeding					
1.	RR	x	x	(X)	o	o	o	o	x	x
2.	JW	o	(X)	x	o	o	o	o	o	x
3.	FC	o	o	(X)	o	o	o	o	x	x
4.	EF	o	(X)	o	o	o	o	o	o	x
5.	LN	x	o	(X)	o	o	x	o	x	x
					(6 out of 26 cases, one without autopsy)					

*Hemorr—hemorrhages; Ca—carcinoma; Intra-H—intrahepatic cholangiolitis; Peri-H—perihepatitis; Fatty—fatty infiltration or (X) degeneration chief diagnosis; Cirr—cirrhosis; Chol—cholangitis; x—minor findings at autopsy; †—inflammatory stenosis.

probing of the common duct, and possibly after operative biliary tree lipiodol x-ray films), suggests the presence of carcinoma or cirrhosis of the liver?

CONCLUSION

In conclusion, we feel that the above evidence lends support to the hypothesis of Ivy, McNealy, and others, regarding the cause of bleeding in obstructive jaundice and to their observations that this bleeding tendency can be prevented or corrected, when permanent liver vitamin insufficiency does not exist, by the administration of an excess of fat soluble vitamins in the form of viosterol. Viosterol administration has a definite prognostic value when correlated with the Ivy bleeding time. Our own observations indicate that this liver vitamin deplention depends upon the duration and the degree of the jaundice, and they stress the value of a careful history in this regard. Finally, we have found the Ivy bleeding time determination a simple and practical clinical test for the detecting of latent bleeding tendencies and for prognosticating about the bleeding course in cases of obstructive jaundice.

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SPLENECTOMY

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ALTHOUGH splenectomy was done in at least a few instances before the birth of Christ,[13] Spencer Wells, in 1865, was probably the first to perform the operation in modern times.[25] In the early years the mortality was high so that the operation was seldom performed. Within the last quarter of a century, however, splenectomy has become a well-recognized and justified procedure. While today it is not to be entrusted to the occasional operator, yet in the hands of a well-trained surgeon the mortality of splenectomy approaches that of any other upper abdominal surgery and depends largely upon the type of cases selected for operation.

The vast majority of patients requiring splenectomy suffer from one of three diseases—splenic anemia (Banti's disease), hemolytic jaundice, or purpura hemorrhagica. There are, however, a number of less common conditions which require removal of the spleen. Splenectomy will be discussed in its relationship to these various diseases and fifty-three cases will be reported. The cases in this series have been divided according to diagnosis in Table I.

Table I

Splenectomy Cases

	DISEASE	NO. CASES	DIED
1	Splenic anemia (Banti disease)	21	8
2	Hemolytic jaundice	12	0
3	Purpura hemorrhagica	7	0
4	Ruptured spleen	5	0
5	Subacute bacterial endocarditis with splenomegaly	2	1
6	Multiple lymphogenous cysts	2	0
7	Syphilitic splenomegaly	1	0
8	Primary tuberculosis of the spleen	1	0
9	Myelogenous leukemia	1	0
10	Aplastic anemia	1	1
		53	10 (18.8%)

Splenic anemia—(Banti's disease)

In 1894 and again in 1910, Banti described a syndrome which has since been associated with his name. He suggested splenectomy as the treatment of choice.[37] The syndrome is characterized by a progressive enlargement of the spleen associated with a persistent anemia. There is a marked tendency toward gastrointestinal hemorrhage. In the terminal stage there is cirrhosis of the liver followed by ascites and

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TABLE II

SPLENIC ANEMIA—21 CASES

NO.	NAME	SEX	AGE	LIVER DAMAGE		PRETHERAPEUTIC HEMOGLOBIN	FOLLOWED	RESULT
				CLINICALLY	AT OPERATION			
1	P. B.	M	40	No	No	45	–	Died, bronchopneumonia
2	E. D.	F	49	Jaundice	Severe	43	–	Died
3	P. L.	M	49	Ascites	Severe	72	–	Died, gastric hemorrhage
4	L. S.	M	42*	Jaundice and ascites	Severe	–	–	Died, gastric hemorrhage
5	A. B.	F	14	Ascites	Severe	–	–	Died, gastric hemorrhage
6	J. C.	M	24	Jaundice and ascites	Severe	80	–	Died, "liver insufficiency"
7	J. L.	M	34	Jaundice	Severe	78	–	Died, "liver insufficiency"
8	P. C.	M	52†	No	Severe	48	–	Died, "liver insufficiency
9	P. C.	F	62	No	Enlarged	55	11 yr.	Well
10	B. S.	F	13	No	–	70	7 yr.	Hemoptosis before and after operation
11	J. G.	M	15	No	–	42	7 yr.	Hemoptosis before and for 4 yr. after operation; now well
12	C. B.	M	35	No	–	68	4 yr.	Well, still moderate anemia
13	L. D.	F	50	No	–	?	5 yr.	Well, Hb. 96%
14	M. S.	F	9	No	–	20%	5 yr.	Well, Hb. 94%
15	H. K.	F	20	No	Severe	76	3 mo.	Symptomatically improved, Hb. 76%; platelets, 170,000
16	S. F.	M	45	No	–	–	1 yr.	Well, Hb. 76%
17	J. K.	M	23*	Ascites	Severe	–	–	
18	J. S.	M	8	No	–	47	3 yr.	Well, Hb. 96%
19	J. F.	F	62	No	Severe	72	7 mo.	Improved
20	W. C.	M	55	No	Liver enlarged	55	3 mo.	Improved
21	E. S.	F	8		Liver enlarged	–	0	

*Omentopexy done in addition to splenectomy.
†Ligation of left gastroepiploic artery.

perhaps jaundice. Very little had been added to the knowledge of the true nature of the disease since Banti's descriptions so that the diagnosis today is still largely one of exclusion. Because of this fact, it is undoubtedly true that while the patients grouped under the diagnosis of Banti's disease present common characteristics there may be different etiologic factors involved.[2, 37, 38]

The treatment of choice in this syndrome continues to be splenectomy. Many feel that splenectomy is worth while only in the early case and that patients who present signs of severe liver damage should not be subjected to the procedure.[4] It has been suggested that ligation of the splenic artery be used in these late cases and good results have been reported.[47] Omentopexy has been done along with splenectomy in those

cases having ascites. Because gastrointestinal hemorrhage is a major threat in many instances, the coronary vein has been ligated at the time of splenectomy.

The series of cases reported from the Mayo Clinic in 1931 included 167 cases with a mortality of 9.6 per cent. Eighty of these were still living, while 63 were well. Of those already dead, 10 lived for more than nine years. The longest survival was nineteen years. Such results certainly seem to justify the operation. Gastrointestinal hemorrhage was the direct cause of death in one-third of the patients who died after leaving the hospital.[13, 32]

Because of the marked difference in the operability of a patient in the early stages and a patient in the late stages of Banti's disease, it is obvious that the mortality can be regulated by the selection of the cases. There were 21 patients in this series diagnosed as Banti's disease. Seven of the group showed evidence of severe liver damage before operation; 6 of these 7 patients died. Of the 14 patients who did not show evidence of severe liver damage before operation, only 2 died. Both of these patients were found to have severely damaged livers at operation. Thus there were 8 deaths in this series; 3 caused by gastric hemorrhage, 1 by bronchopneumonia, while in 4 instances the cause was unknown. These last were spoken of as "liver deaths."

The results following splenectomy for splenic anemia depend to a great degree upon the stage of the lesion. In Table II may be seen the results of the cases in this series. The longest survival to date is eleven years. There are three patients perfectly well, with over 90 per cent hemoglobin from three to five years after operation.

HEMOLYTIC JAUNDICE

Hemolytic jaundice has long been divided into the congenital and acquired forms. There is some doubt, however, that the disease is ever acquired.[45] When the disease is first noticed after childhood and is not associated with a family history, it is usually spoken of as acquired. Under such circumstances the disease is frequently most severe.[48]

When normal red blood cells are placed in hypotonic salt solution, they become more globular as they increase in volume and finally rupture. The cells in hemolytic jaundice are already partially changed in that direction. Thus Haden[15] has demonstrated spherocytosis which causes the cells to have a smaller diameter (microcystosis), whereas, the volume is normal or increased. This phenomenon explains in part the characteristic increase in red blood cell fragility found in this disease.

In addition to these findings, there is usually a greenish pallor to the skin, with an increased Vandenberg reaction. There is a normal or increased amount of bile in the stool. There are abnormal quantities of urobilin and bilirubin in the urine. There is a secondary anemia with its accompanying increased reticulocyte count. The spleen is enlarged and usually palpable.[5]

TABLE III

HEMOLYTIC JAUNDICE

NO.	NAME	SEX	AGE	DURATION OF SYMPTOMS	FRAGILITY	PEROPERATIVE HB.	F. U.*	GENERAL CONDITION	BLOOD
1	G. M.	M	17	8 yr.	.525-.400		2 yr.	Well	Normal
2	B. M.	F	18	3 yr.	.625-.400		3 mo.	Well	
3	E. H.	M	20	2 yr.	.725-.425		2 yr.	Well	Normal Hb. 100%
4	L. M.	F	28	--	.750-.425	33%	1 yr.	Well	R.B.C. 4,300,000 Hb.—82% P.—148,000
5	T. L.	M	30	3 yr.	.500-.400	30%	1 yr.	Unimproved, continued jaundiced	Jaundiced
6	M. W.	F	56	--	.500-.400		1 yr.	Well	Hb.—80% P.—normal
7	C. L.	M	22	6 yr.	?		1 yr.	Well	
8	A. M.	F	4	4 yr.	.675-.400		3 yr.	Well	
9	R. M.	M	9	5 yr.	.650-.450	22%	3 yr.	Well	
10	E. M.	F	11	1 yr.	.625-.375	70%	3 yr.	Well	
11	N. G.	M	20 mo.		.625-.450	63%	Recent		
12	E. S.	M	18	6 yr.	.525-.400	--	-		

*Follow-up examination.

Splenectomy was first performed for hemolytic jaundice by Spencer Wells in 1887. The patient was still alive when last reported in 1932.[4] This operation, used empirically and found to be successful, has stood the test of time. When the diagnosis is certain, a complete and permanent cure can be expected to follow splenectomy. In the atypical case, the result may be unsatisfactory.[44]

It has been pointed out that although in a state of chronic anemia due to continued hemolysis, many of these cases go along for years moderately comfortable, so that one should consider the surgical mortality before advising operation.[17] Since the operative mortality in these chronic cases has been reduced to a minimum, it is felt that splenectomy should be advised in all typical cases unless contraindicated.

In the 12 cases in this series there were no deaths. In the 118 cases reported from the Mayo Clinic, the mortality was only 3.4 per cent.[32] A mortality in this neighborhood can generally be expected in chronic cases. The results following operation frequently border on the dramatic. The jaundice may disappear in a few days and the blood count may return to normal in a few weeks. The red blood cell fragility may approach but never return to normal.

Of the 12 cases in this series, only 1 has been a failure. It was recognized as being atypical at the time of operation. This patient has since been reported in detail by Hamburger and Bernstein[16] as a case of chronic hemolytic anemia with paroxysmal nocturnal hemoglobinuria. In about one-half of the patients in this series, the follow-up period has been quite short. The results are given in Table III.

The reports in the literature are good in a high percentage of cases. Several authors[10] have reported cases with recurrent jaundice. It is probable that these were cases atypical in type or with accessory spleens. In the Mayo Clinic series[32] of 98 cases, 82 were cured, 12 improved, and 4 unchanged. It is the consensus of opinion that if the patient presents a typical picture of hemolytic jaundice, one may expect complete and permanent relief in a high percentage of cases following splenectomy.

It has long been the teaching that splenectomy should not be performed during an acute exacerbation of the disease. All agree that it is safer to operate during a quiescent period. However, the acute exacerbation may be so severe that one is forced into some life-saving measure. Under such conditions, a blood transfusion is frequently hemolyzed regardless of the care of cross agglutination tests. Because of the fear of posthemolytic anuria, transfusions should be reserved until after splenectomy.

Doan, Curtis, and Wiseman[6] believe that splenectomy is the procedure of choice in the acute hemoclastic crisis, whether it is spontaneous or due to some precipitating factor. They were forced into operation in 5 acute cases when the red cell count was in the neighborhood of one million. They found that the red cell count rose about one million while the patient was on the table. Studies in blood volume led them to believe that this was due to a contraction of the spleen.

PURPURA HEMORRHAGICA

Splenectomy was first suggested for purpura hemorrhagica in 1916 by Kaznelson.[19] The results in his cases were so remarkable that the operation was immediately adopted by most surgeons. Although the spleen has not been definitely proved to be the seat of the disease in purpura hemorrhagica, its removal has been followed by results justifying the continuation of the procedure on an empiric basis.

The establishing of a positive diagnosis is of paramount importance before splenectomy is considered. The disease is characterized by (1) petechial ecchymosis and bleeding from the mucous membranes, (2) low platelet count, (3) prolonged bleeding time, (4) normal coagulation time, (5) delayed or absent clot retraction, (6) a secondary anemia with evidence of regeneration, (7) a normal or increased total white count with a normal differential count, (8) a tourniquet test that may be positive, (9) the spleen that may or may not be palpable. Errors in diagnosis are most frequently made in cases of acute leucemia or aplastic anemia associated with hemorrhagic phenomenon.

Purpura hemorrhagica as it confronts the clinician may be divided into four groups:

Group 1, Mild or Moderate Bleeding in the Initial Attack.—There is probably universal agreement that patients in this group should be

TABLE IV

PURPURA HEMORRHAGICA

NO.	NAME	SEX	AGE	ATTACKS	SEVERITY	DURATION	PREOPERATIVE BLOOD	F.U.	CONDITION AND BLOOD
1	M. T.	M	20	Repeated	Mild	10 yr.	P—12,800	5 yr.	
2	R. V.	M	2	Initial	Moderately severe	2 wk.	P—12,000	4 yr.	No recurrence, Hb. 94%, P—448,000
3	E. S.	F	6	Initial	Moderately severe	3 wk.	P—9,600	1 yr.	No symptoms, Hb. 76%, P—50,000
4	G. B.	F	19	Repeated	Severe exacerbation	6 mo.	P—17,600 Hb.—32%	1 yr.	No symptoms, Hb. 86%, P—250,000
5	J. R.	M	20	Repeated	Moderate	1 yr.	P—7,000 Hb. 64	2 yr.	Well, Hb. 96%
6	J. S.	M	11	Recurrent		9 yr.	Hb. 45	No F.U.	
7	C. L.	F	11	Initial attack	Severe	4 da.	Hb. 38 P—12,000	No F.U.	

treated conservatively by adequate diet, elimination of foci of infection, and transfusions if necessary. Under such treatment the majority will get well and stay well.[14]

Group 2, Mild or Moderate Bleeding in a Recurrent Attack.—There is almost universal agreement that patients in this group, having given no response to conservative therapy, should be subjected to splenectomy.[8, 42, 44, 48] Some feel that splenectomy should be reserved for the fulminating case with uncontrollable hemorrhage, or for the recurrent case sufficiently severe to interfere with normal growth.[24]

Group 3, Severe Bleeding in the Initial Attack.—The opinion differs as to the treatment of patients in this group. On the basis of collected cases, Whipple[48] and Spence[42] advise against splenectomy in the acute attack. The mortality in the 35 cases reported until 1932 was 34.3 per cent.[7] This, however, was due largely to the high mortality in the early cases. In the last 21 cases after experience had shown that great care had to be taken as to the pre- and postoperative treatment, the mortality was only 13.6 per cent. Pemberton[33] probably expresses the feeling of the majority when he points out that if the bleeding cannot be controlled by packing and transfusions, early operation is indicated. The high mortality in the acute cases will be found in those cases where operation has been postponed until one is forced into splenectomy as a last resort.

Group 4, Severe Bleeding in a Recurrent Attack.—These patients have previously demonstrated their ability to go into remission. For this reason they should be treated by transfusion and given every opportunity to again go into remission. However, the patient must not be allowed to get in poor condition before the decision is made to resort to splenectomy.

Many nonoperative forms of treatment have been suggested for purpura hemorrhagica, such as a high vitamin diet,[13, 18] thromboplastic injections,[18] antivenom injections,[12, 30, 43] nonspecific protein shock,[9, 28] and ovarian extract in menstrual purpura.[21] Although x-ray therapy has received relatively little encouragement,[29] excellent results have recently been reported in a short series of cases.[39]

The results following splenectomy for purpura hemorrhagica have been sufficiently satisfactory so that one is justified in advising operation in any case where the disease is an inconvenience and has not responded to conservative measures. According to the published reports up to 1932,[7] 87 per cent of 179 followed cases were cured. There was, however, an admittedly short follow-up in many instances. In the Mayo Clinic series[33] with long follow-up periods, 63 per cent were permanently cured. In only one instance, however, was the recurrent bleeding of a serious nature. Other reports continue to be favorable in a high percentage of cases.[1, 35, 45] Griffin,[13] with a large experience, states that he has never seen purpura persist after splenectomy and a conscientious elimination of foci of infection. It has been suggested[26] that accessory spleen may account for many cases of apparent cures followed by recurrence.

The 7 cases of purpura hemorrhagica reported in this series are shown in Table IV. On an incomplete follow up only 1 patient had had any recurrent bleeding and this consisted of 2 insignificant nose bleeds in 5 years.

RUPTURED SPLEEN

Rupture of the spleen is usually the result of violent trauma. It occasionally occurs spontaneously.[52] When the diagnosis is made, it is safest to operate as soon as the patient is in condition, even though the hemorrhage has apparently stopped, because of the danger of delayed or recurrent hemorrhage.[23]

In this series there were 5 cases of ruptured spleen, all due to severe trauma. The patients have been followed from one to eleven years. All of these patients are perfectly well and have noticed no ill effects from the loss of the spleen. The cases are given in Table V.

TABLE V

RUPTURED SPLEEN

NO.	NAME	AGE	FOLLOW-UP	GENERAL HEALTH	BLOOD STUDIES
1	E. E.	25	11 yr.	Excellent	R.B.C.—4,800,000 Hb.—90%
2	R. S.	6	4 yr.	Excellent	R.B.C.—4,000,000 Hb.—72% P—576,000
3	E. R.	14	1 yr.	Excellent	--
4	A. D.	12	5 yr.	Excellent	--
5	M. B.	55	2 yr.	Good	--

Removal of the normal mammalian spleen was found by Krumbhaar[20] to cause a mild temporary anemia, with an increase in the resistance of the red blood cells. Gottlieb[11] has advanced the theory that the spleen is normally active in iron metabolism so that the iron is not retained and utilized following its sudden removal. Thus a temporary anemia is produced until the other parts of the reticuloendothelial system gradually take over this function of the spleen. He was able to decrease the severity of this transitory anemia by feeding iron.

UNCOMMON CONDITIONS

There are several other conditions for which a patient is occasionally subjected to splenectomy which happen to be represented in this series.

Splenectomy was performed twice in this series upon patients with subacute bacterial endocarditis with splenomegaly. One had a splenic abscess. This patient died one week after operation from a cerebral hemorrhage. The other patient was not followed. Riesman, Kolmer, and Polowe[36] have reported 4 cases of splenectomy for subacute bacterial endocarditis and feel that life is prolonged and made more comfortable by the operation.

Multiple lymphagenous cysts of the spleen was the diagnosis in two cases. Both of these cases have completely recovered and have normal blood counts two and four years after operation. In one instance, a 3,400 gm. spleen was removed from a 100 pound, twenty-year-old woman.

Splenomegaly is occasionally associated with a case refractory to antisyphilitic treatment. Splenectomy is then indicated as a part of the antisyphilitic treatment. One such case was included in this series, a seventeen-year-old boy with congenital syphilis. Following splenectomy he responded satisfactorily to treatment.

Primary tuberculosis of the spleen has been reported in 51 instances up until 1912 according to Winternitz.[50] Since that time a somewhat smaller number have been added to the literature. A positive preoperative diagnosis has been made only in those cases where calcification of the spleen was demonstrated by x-ray.[41] Isolated healed tubercules have been found in the spleen on autopsy, but such spleens were normal in size. When a primary tuberculosis of the spleen has assumed the magnitude of a clinical splenomegaly, the prognosis is probably hopeless without surgery. The diagnosis is to be considered in a case of splenomegaly of unknown origin with fever, loss of weight, and lassitude. There may be a close similarity to splenic anemia.[3, 34, 40] When splenectomy is done before other tuberculous foci become established, the results are uniformly good. In our case, the patient, aged fifty-one years, had lost forty pounds in weight in eighteen months. The spleen weighed 2,100 gm. All other studies were negative, except for a moderate anemia. Following splenectomy the patient

regained his normal weight and has been perfectly well for two years. His red cell count is now 4,800,000 and his hemoglobin is 98 per cent.

Splenectomy was done for myelogenous leucemia only once in this series. The patient died three months after operation. Griffin[13] feels that if there is no evidence of acute leucemia, if the history is short, and if there is no marked anemia, splenectomy is justifiable after preoperative irradiation. In the 43 cases reported by him, 20 were well for two years, while 3 were well over five years.

This series contains one case of aplastic anemia that was associated with purpura. Splenectomy was done as a last resort after six months of conservative treatment. There was no response of the bone marrow, and the patient died on the eighth postoperative day.

TECHNIQUE

The operation of splenectomy may be a very difficult one or a relatively simple one, depending upon the size of the spleen and the presence or absence of adhesions. The left rectus incision is probably most frequently used. The lower pole of the spleen is delivered first and brought out of the wound. The upper pole of the spleen then may be exposed. By using this procedure, the size of the incision need not be so large as would otherwise be required. When the spleen is very large, it may occasionally become necessary to make a lateral transverse extension of the incision at the lower end. This gives considerable additional exposure. Such a step is seldom required but may be a valuable aid when an especially large spleen is encountered. Some surgeons find the left paramedian[49] or the left subcostal[31] incision preferable to the left rectus incision.

By means of the gloved finger, the spleen is freed posteriorly. In this manner the capsule is not torn. The lienorenal ligament may occasionally have to be nicked with an instrument. The lower pole of the spleen is separated first and brought out of the incision. As this is done, the upper part of the spleen can be exposed. When the posterior attachment has been completely freed, the spleen is turned up to expose the posterior aspect of the pedicle devoid of a peritoneal covering. The peritoneum is then slit over the anterior aspect of the pedicle and the splenic vessels are exposed. Care must be taken that the tail of the pancreas, the stomach, and the retroperitoneal duodenum are sufficiently pushed aside before hemostats are applied. The vessels are clamped close to the spleen, which is then removed. The vessels are doubly ligated. Wise[51] has suggested the use of a rubber-shod clamp across the entire pedicle until the vessels are ligated individually. It would seem that he has minimized the possibility of damage to the pancreas by this procedure. The oozing from the bed of the spleen is usually so insignificant that no drainage is necessary. When the

ooze is sufficient, a Penrose drain may be inserted. The drain is best brought out through a stab wound lateral to the incision.

When a large spleen has been removed, an abdominal binder is applied immediately to help maintain abdominal pressure. Pitressin is given every fourth hour for the first forty-eight hours. The convalescence may be marked by a moderately febrile course which is frequently quite prolonged.[27] This is probably due to a thrombophlebitis of the splenic vein as mentioned by Bryce.[2] No deaths due to extension of the thrombosis into the portal vein have been recognized in this series.

SUMMARY

1. A series of 53 splenectomies is reported, with a gross mortality of 18.8 per cent. The late cases of Banti disease with clinical evidence of severe liver damage (ascites or jaundice) were responsible for the greater part of the mortality. If these cases were eliminated from the series, the mortality would be 8.6 per cent. There were no deaths in 19 cases of hemolytic jaundice and purpura hemorrhagica.

2. The results to be expected in the various conditions following splenectomy are discussed.

3. A case of primary tuberculosis of the spleen is reported.

4. The technique of splenectomy is discussed.

CONCLUSIONS

1. Splenectomy in splenic anemia (Banti disease) had best be confined to the early stages. Splenectomy is justified in the late stages only when the discomfort due to the size of the spleen makes life miserable for the patient.

2. Splenectomy is the treatment of choice in hemolytic jaundice. It is preferable to perform the operation during a remission, but occasionally it is necessary to remove the spleen during an acute exacerbation. The results following splenectomy are good in a high percentage of cases.

3. Cases of chronic hemorrhagic purpura that do not respond to conservative therapy should be subjected to splenectomy. In acute purpura hemorrhagica, if the bleeding does not respond promptly to conservative measures, splenectomy should be performed early before the patient's condition is critical. A careful elimination of infection is important in the prevention of recurrent purpura. Recurrence of a serious nature is unusual.

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ABNORMAL PROMINENCE OF THE EARS: A METHOD OF READJUSTMENT

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INTRODUCTION

AT ONE time or another, every surgeon is asked what can be done to restore projecting ears to normal position. This problem is one that quite frequently has been brought to us for solution, and we have found that the solution is not quite as simple as it may seem at first glance. In addition to the correction of the actual deformity, we have to deal with the psychologic aspect of the question, both from the standpoint of the patient, and frequently from that of the parents as well. For this reason in children, we often replace congenitally prominent ears into normal position before they begin school in order to avoid the distressing mental effect caused in so many instances by the gibes and cruel remarks of the other children.

We have in our series a number of cases where the replacement of prominent ears has wrought a complete change in the mental complex of the child and has been the beginning of a new and happy life.

In older patients, the results for the better are sometimes psychologically astounding, since many of them had been unaware that anything could be done to correct the deformity and had gone on in life trying to hide their feelings, or had appeared in public as infrequently as possible.

There is a group, however, who are unaffected psychologically, but who come for help principally for practical reasons: aviators who cannot comfortably wear their snug helmets; young women who are unable to wear the tight hats which the fashion of the moment may call for. To this group may be added those who cannot get suitable employment on account of the odd appearance caused by widely outstanding ears.

The external ear is formed during the sixth week of fetal life from six tubercles which appear on the mandibular and hyoid arches around the first cleft depression and also from the elevation of the skin behind the tubercles on the hyoid arch. The ear begins to assume a definite form during the latter part of the second and the first part of the third month. The margin of the ear at the posterior superior angle is pointed (Darwin's tubercle) and because the crura of the antihelix are unformed, the ear protrudes from the head. About the sixth fetal month, the margin curls, forming the helix, the antihelix becomes more definitely folded,

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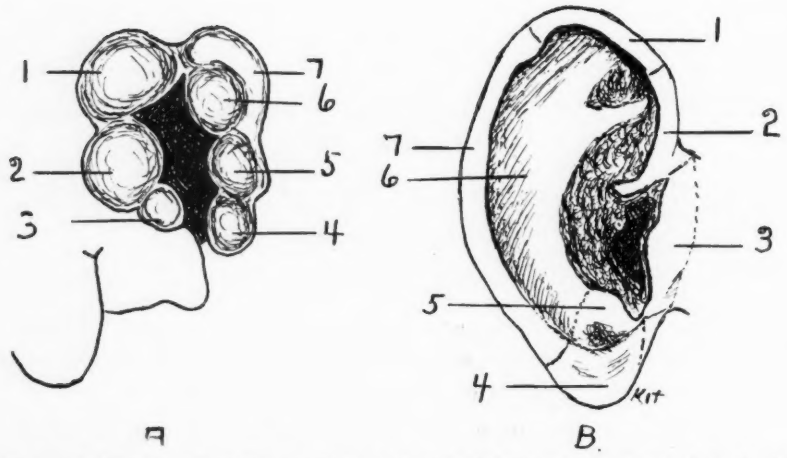


Fig. 1.—Schematic drawings to show the embryology of the ear. *A 1, 2, 3,* Tubercles which arise from the first mandibular arch to form the tragus, *B 3;* crus of the helix, *B 2;* and helix, *B 1.* *A 4, 5, 6,* Tubercles which arise from the hyoid arch to form the lobe, *B 4;* antitragus, *B 5;* antihelix *B 6.* *A 7,* Skin elevation behind the tubercles of the hyoid arch which form the descending helix or posterior margin of the ear *B 7.*

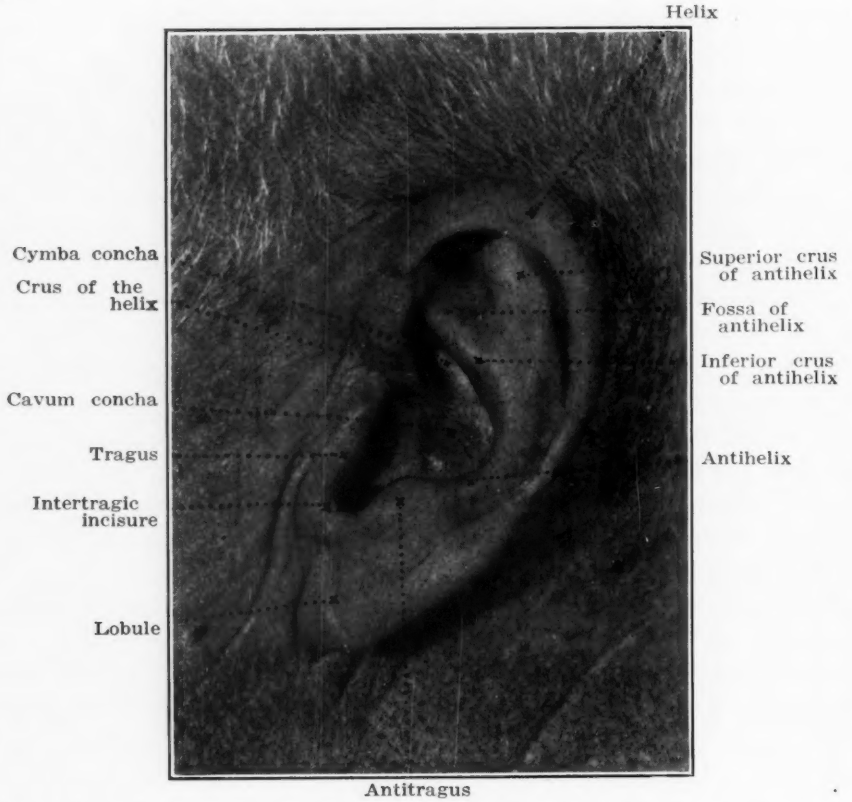


Fig. 2.—Photograph of an ear in normal position with a well-developed antihelix and its crura. Note the strong supporting ridges.

and its crura appear. When completely formed, the normal ear rests at an angle of about 30° to the head. The lower portion of the antihelix is usually well developed; in some instances the superior crus is present; and in some both crura are present, but the folding of the cartilage

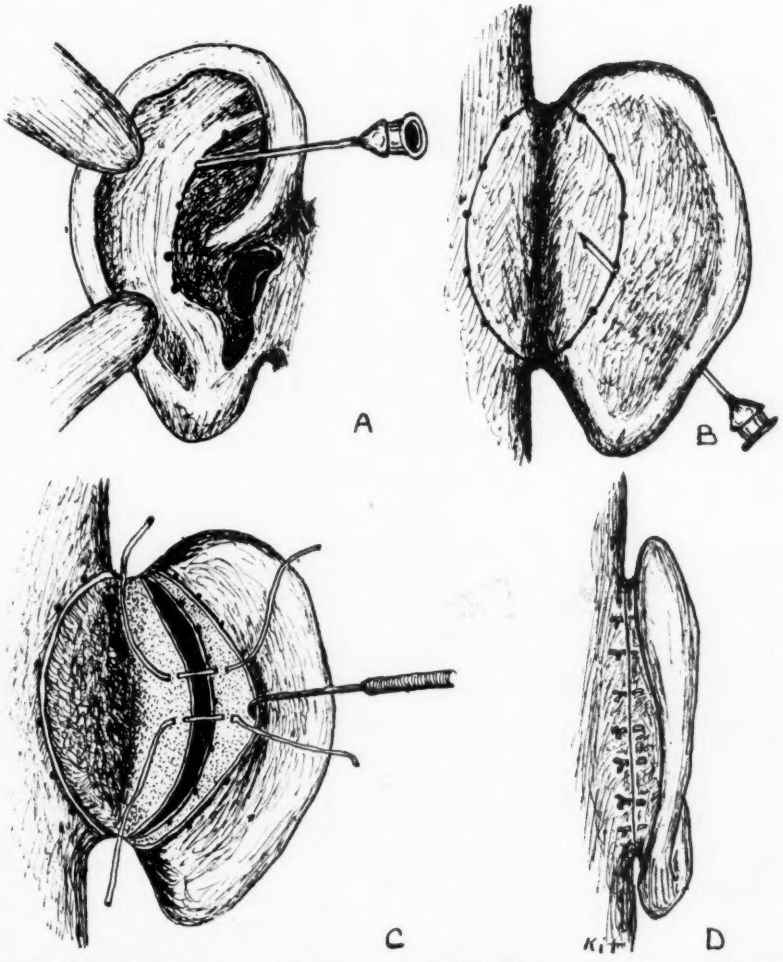
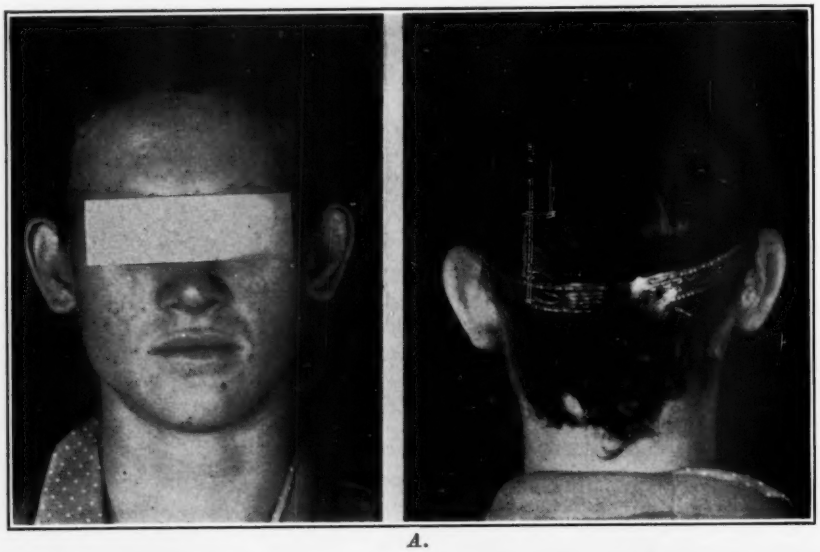
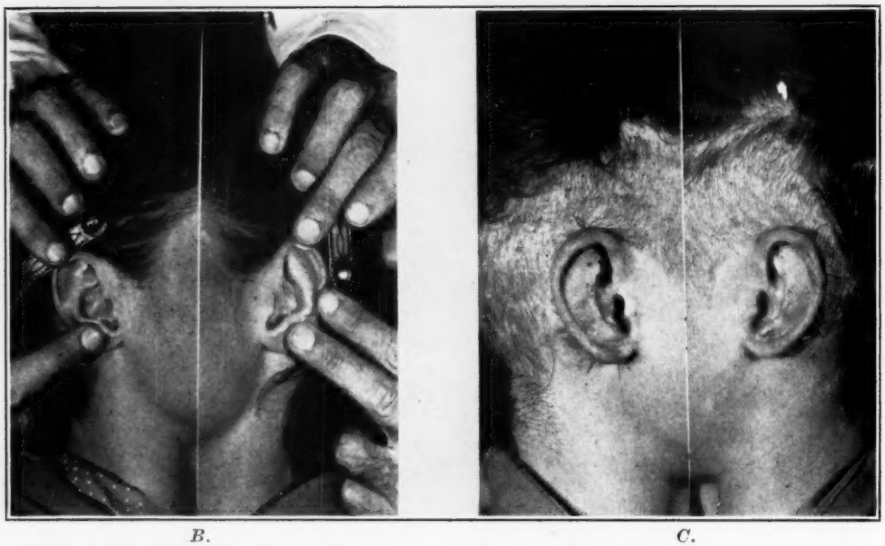


Fig. 3.—Schematic drawings showing the steps in the operation for the replacement of prominent ears. *A*, The ear is placed in its normal position against the head and the upper and posterior limits are marked on the scalp with an hypodermic needle dipped in brilliant green, 5 per cent in alcohol. Note two punctures above the ear and one behind. With the ear in this position the undeveloped antihelix will reform sufficiently to indicate the line of proposed reconstruction. Along this line with an hypodermic needle dipped in the brilliant green solution, punctures are made completely through the ear and about 1 cm. apart. *B*, Shows the puncture marks and the needle emerging on the posterior surface of the ear through one of them. The needle shown in the drawing is comparatively much larger than that actually used which is 24 gauge and 1 inch long. The points of perforation are connected with a line of brilliant green and while the solution is still damp the ear is pressed against the head and a contact line is made. This line is freshened with the green solution and the ends are carried forward and joined, making an elliptical-shaped pattern. The area of skin thus outlined is that which is to be removed. *C*, The skin has been removed. The marks of the perforations in the skin can be seen. The second line of green-stained dots are those left in the cartilage by the needle punctures through the ear. The area from which the cartilage was excised, which conforms to the curve of these perforations, is shown. The cartilage spring has been broken. Two sutures of catgut of the type used to turn in the cartilage edges to form the antihelix can be seen placed in the perichondrium. *D*, The ear assumes normal position and the skin is closed with on-end mattress sutures of horsehair.

has not been sufficient to support the ear normally close to the head. In some instances, both crura are completely unfolded and give no support at all.



A.



B. *C.*

Fig. 4.—Illustrating the ordinary type of congenital bilateral prominent ears, seventeen-year-old girl. Marked mental distress. *A*, The buttressing folds of the antihelix are undeveloped and the crura are unformed on each ear. *B*, With the ear held against the head, the antihelix reforms sufficiently to show the ridges to be reconstructed. *C*, Twelve days after reconstruction of the antihelices. The puncture marks stained by the brilliant green are still present in the skin, and the ends of the horsehair sutures can be seen projecting beyond the ear margins. *D*, Two weeks after operation. Note the difference in the position of the ears following the reconstruction of the antihelices. Since the ears were restored to normal position, the mental attitude of the patient has been vastly improved.

Prominent ears are fairly common. The reason that the cartilage does not fold into the normal, buttressing ridges in these cases is not

known. There is normally a sharp ridge of cartilage between the concha and the fossa above and behind the antihelix, and if this supporting ridge is missing, the deformity results. In other words, in the majority of cases the incomplete development of the antihelix, especially of the crura, is the principal cause of the projecting ears.

Abnormal prominence of the ears is usually congenital, especially in the marked cases, but it is possible that they may be acquired by young children constantly sleeping with the ear curled up, or by having the ears frequently rolled forward under carelessly placed caps.

We have noted in a number of instances that heredity seemed to be a definite factor. The deformity is usually bilateral, but may be unilateral. Sometimes the prominence is more marked on one side than on the other,

Fig. 4*D*.—See opposite page for legend.

and the actual deformity of each ear varies. Sometimes the ears are of different sizes and are not on the same level. Ordinarily hearing is not affected by the prominence of the ears.

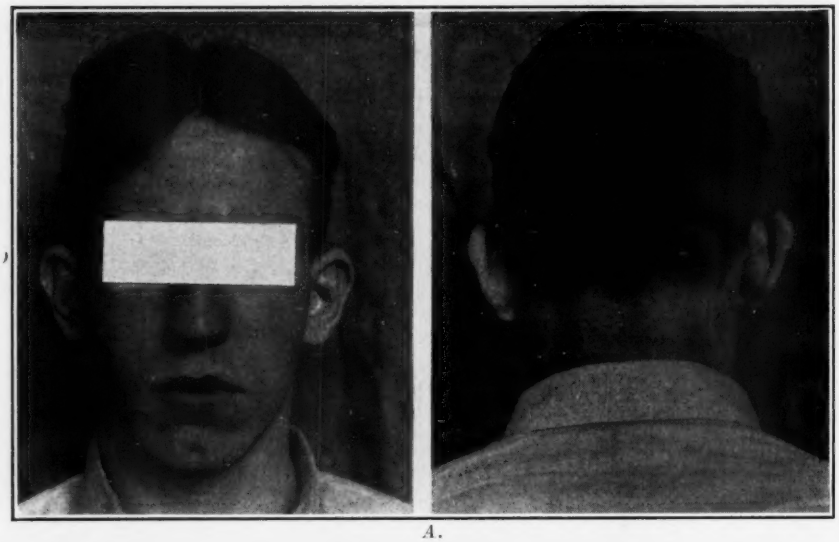
Abnormally prominent ears may be divided into two general groups: first, where the ear itself is of normal shape but is attached to the head in an abnormal position; second, where the prominence is due to an abnormality of the ear itself. In both of these types the ears may be enlarged, but in the second group we frequently find that when the unfolded cartilage is readjusted, the ear which was apparently enlarged assumes the normal size.

We also find in the first group that ears which seem much too large when in the abnormal position are inconspicuous in regard to size when the angle is changed and when they are placed closer to the skull. Con-

sequently correction of the macrotia is unnecessary. It is seldom that prominent ears are smaller than normal.

In congenitally prominent ears, we find that the cartilage of the ear is sometimes soft and floppy. Ordinarily, however, the cartilage has normal rigidity.

There have been a number of operations suggested for the correction of prominent ears. These vary from the simple excision of an ellipse of

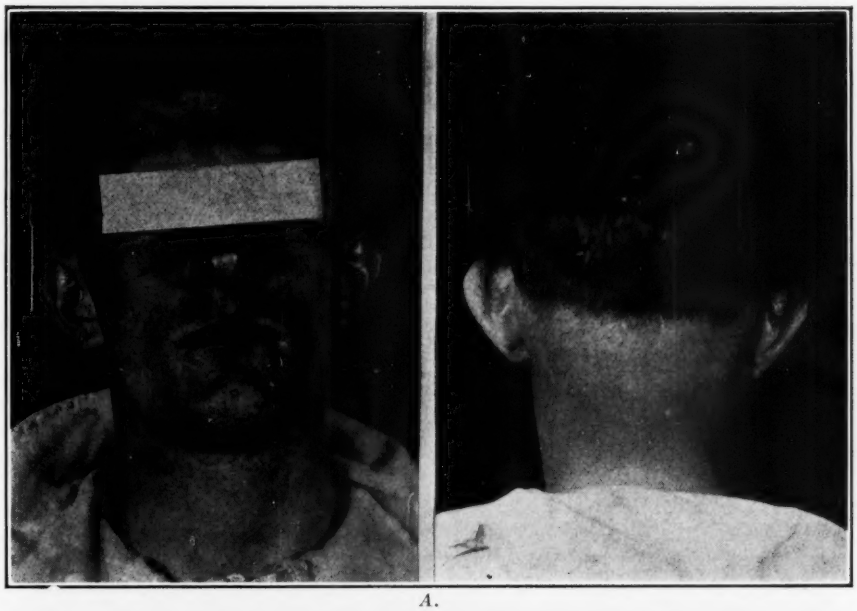


A.

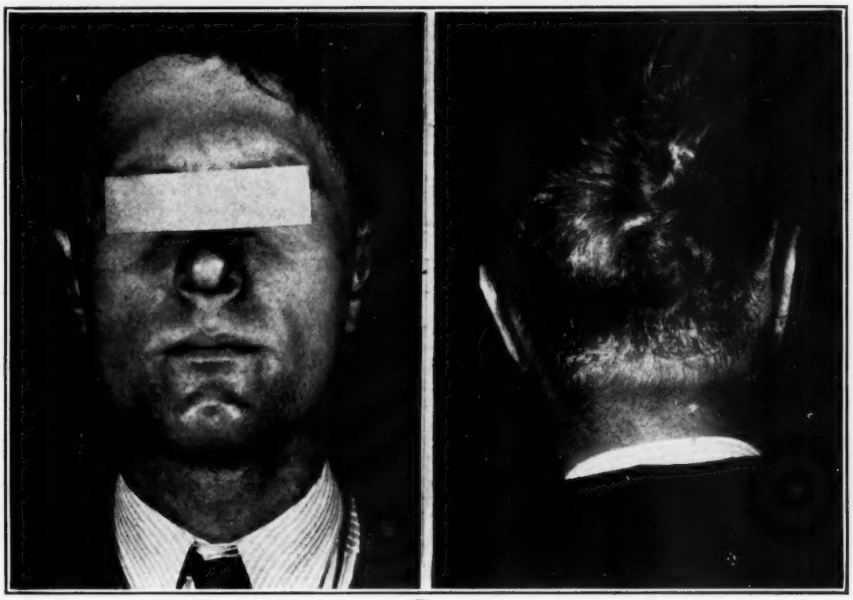
B.

Fig. 5.—Illustrating a moderate grade of congenital bilateral prominence of ears, nineteen-year-old male. Marked mental distress. Complains of inability to get a job. *A*, The antihelices are incompletely developed. Note the position of the ears. *B*, Result after seven years. The ears are not quite as close to the head as they were immediately after the overcorrection operation, but there has been no change in position after the first few months. The mental disturbance caused by the prominence of the ears cleared up soon after operation.

skin with subsequent closure to more complicated procedures in which the cartilaginous framework of the ear is involved. Excision of skin alone should be considered only in cases of slight deformity in young children, or where the cartilage is very soft, and even in these cases the ultimate result is uncertain. If we expect to get a lasting result, it is



A.

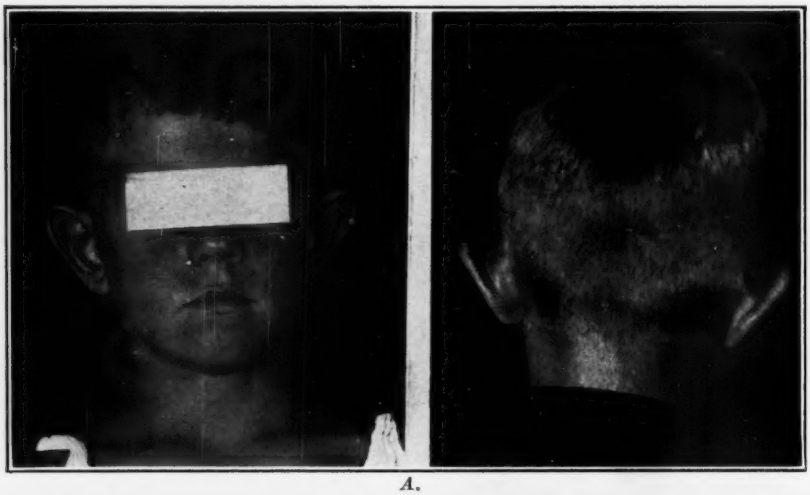


B.

Fig. 6.—Illustrating congenital bilaterial prominence of ears associated with uncurled helix, twenty-four-year-old male. Odd appearance interfered with obtaining suitable employment. *A*, Note the prominence and position of the ears. The crura of the antihelices are undeveloped as is the curl of each helix. The deformity in the right ear differs from that of the left ear. *B*, Result of reconstruction two weeks after operation. Nothing was done to the helix.

necessary to break the spring of the cartilage by some means, whether it is firm or soft, and to reconstruct the buttressing ridges so that the ear will be supported and will remain in the approximate position in which it is placed by the operation.

In order to overcome the resistance or spring of the cartilage, several procedures have been used: thinning the cartilage along the line of fold-



A.

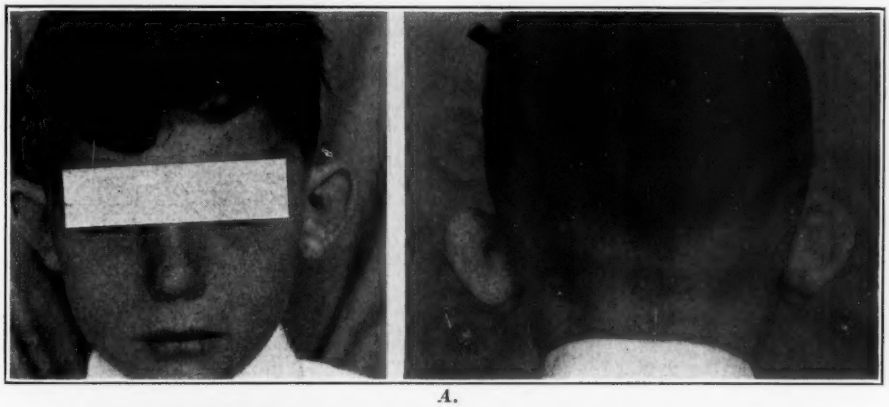
B.

Fig. 7.—Illustrating congenital bilateral prominence of the ears, eight-year-old boy. Constantly in trouble on account of remarks made in school by his companions. *A*, Note the unfolded crura of the antihelices. The upper portion of the ears stands away from the head considerably further than the lower. *B*, Two weeks after the reconstruction of the antihelices. The ears appear somewhat too close to the head, but the retroauricular angle on each side is deep, and the ears will gradually come out slightly.

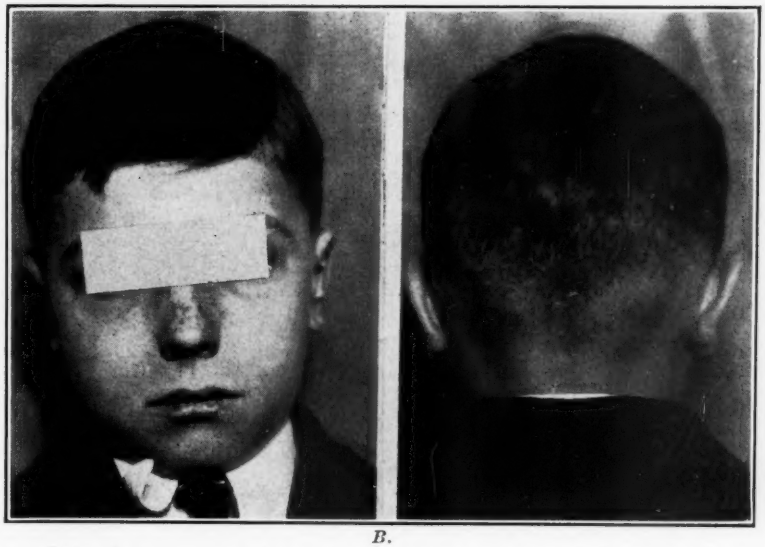
ing; incising the cartilage along this line; excising long narrow crescentic- or elliptical-shaped pieces of the cartilage along the line which is to be turned in; or both excising and incising the cartilage. We will

not attempt at this time to review the various methods used. The purpose of this paper is to describe a technique which we have gradually developed, particularly for those deformities caused by abnormalities of the antihelix, and which we have been using for a number of years with satisfaction.

The method presents several points which have been very useful to us and while they may have been used elsewhere, as far as we can ascertain, they have not been reported in quite the same way that we use them.



A.



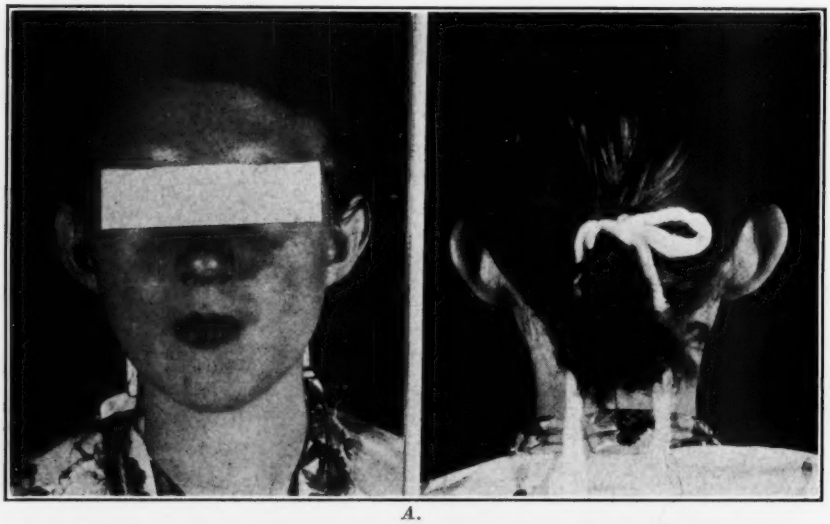
B.

Fig. 8.—Illustrating a marked case of congenital bilateral prominence of the ears, nine-year-old boy. Much mental disturbance caused by the gibes of schoolmates. *A*, There is complete undevelopment of the supporting ridges of the antihelices. The ears stand away from the head almost at right angles. *B*, Three and one-half months after the restoration of the ears to normal position by reforming the antihelices. This patient was seen recently, seven years later, and the ears have stayed in good position. The mental distress ceased immediately after the patient observed his ears at time of first dressing.

METHOD

We prefer a general anesthetic, usually avertin, 60 to 75 mg. per kilogram, supplemented by nitrous oxide and oxygen. The operation

can easily be done under local anesthesia, but it is tedious and we have found that the patient, in the long run, is better off under a general anesthetic. The patient is placed on a brain table with the face down. In this way both ears are fully exposed at the same time in the most



A.

B.

Fig. 9.—Illustrating congenital bilateral prominence of the ears, fourteen-year-old girl. Inferiority complex. *A*, Note the marked deformity and the lack of development, especially of the crura. *B*, Result three months after reconstruction of the antihelices. Note the newly formed supporting ridges and the position of the ears. The inferiority complex has been forgotten. *C*, Result after three and one-half years. Note the normal position of the ears and the permanence of the reconstructed ridges.

favorable operative position, and the clean-up and drape on both sides can be done before the operation is begun. The hair is shaved around the ear for a sufficient distance; the external auditory canal is plugged

with cotton wet with alcohol; and this is followed by a thorough clean-up with ether, alcohol, and kalmarid.

The ear is now held in normal position against the side of the head, and its upper and posterior limits are marked on the scalp with 5 per cent brilliant green in alcohol in order to indicate the general position to be attained by operation. When the ear is in this position, it will be found that the portion of the antihelix which is unfolded will reform itself sufficiently to give the line of required reconstruction, and this line is marked out on the anterior surface of the ear with the brilliant green solution. Then along the full length of this line a series of punctures, five or six or more in number, and about 1 cm. apart are made in the following manner. An hypodermic needle, at least one inch

Fig. 9C.—See opposite page for legend.

long and about 24-gauge, dipped in brilliant green solution and placed at right angles to the anterior skin surface is thrust completely through the ear from front to back, coming out at a corresponding skin point on the posterior surface. When the needle emerges through the skin of the back of the ear, its point is touched with a toothpick swab dipped in the same solution, before it is drawn back again, and in this way sufficient dye is carried through the tissues to make the required marks on the cartilage but not enough to overstain, as would be the case if there were dye in the needle or syringe. A green puncture mark shows on the skin of the anterior surface of the ear where the needles enter and similar marks are found on the skin of the posterior surface of the ear where the needles emerge. The ear is drawn forward, the puncture marks on the back of the ear are connected by a line of brilliant green applied with a toothpick, and this line while damp is pressed against the skin of

the scalp with the ear in normal position. This gives a slight greenish mark which is supplemented to make it more distinct, and the area inside these lines, which when the ends are extended and joined is more or less elliptical in shape, will be the skin area to be excised.

The excision is made, all bleeding is checked and necessary vessels tied. When the cartilage is exposed, a line of puncture points stained green can be seen in the cartilage itself which definitely marks out the direction and curve to be followed. Then, depending on whether an elongated crescent or ellipse of cartilage is to be removed or whether a simple incision through the cartilage is to be made, or whether both incision and excision are used, the selected procedure is carried out with a small sharp blade. We find that the finger in the anterior surface of the ear will give one a guide and will aid in preventing the perforation of the skin on this surface. If an area of cartilage is to be excised, the incision is made all around it and completely through it, and it is then stripped out with a thin flat elevator.

It is important to see that the incision or excision of the cartilage extends the full length of the antihelix and that all cartilage spring is divided so that there will be no resistance or tension when the margins are turned in to form the new antihelix. The new antihelix is formed by turning forward the margins of the cartilage to form a supporting ridge. This is done by passing a catgut suture (No. 0 or No. 1, plain) through the perichondrium on one side beginning about 0.5 cm. from the cartilage margin and coming out close to the margin. The needle is then carried across the defect and enters the perichondrium a similar distance from the margin and then out about 0.5 cm. from the defect. This suture is of the Lembert type. Four or five of these sutures are placed and then all are drawn up at one time and are tied in order. It will be found that the cartilage edges have been turned forward toward the anterior surface of the ear, replacing the unfolded portion of the antihelix, and that the ear immediately assumes a position which approximates normal. The skin is then closed with on-end mattress sutures of horsehair.

After the operation is completed, the ears will sometimes appear to be almost too close to the head, but in time the sharp overcorrection of the reconstructed antihelix will relax.

DRESSINGS

The dressing of the reconstructed ear is of considerable importance, since immobilization and perfect support of the newly adjusted cartilage are essential for a good result. The sutured line is painted with one-third strength tincture of iodine. A single layer of gauze impregnated with 3 per cent xeroform ointment is laid over the stitch line. Over this is placed a narrow strip of sterile seasponge between the ear and the scalp, and the ear is held close to the head. Then with small bits of sea-

sponge every portion of the anterior part of the ear is carefully and snugly packed, eliminating all dead space and giving absolute support to the readjusted cartilage. After the ear has been completely filled with the sponge bits, a single larger piece of seasponge is placed over the whole and secured by strips of adhesive. Over this is placed a snug bandage, care being taken to avoid pressure sloughs. Unless some untoward symptom presents, the dressing is allowed to remain undisturbed for ten days, at which time the horsehair stitches are removed, and a snug dressing is applied. All dressings are completely removed in another week, and the patient is instructed to hold the ears close to the head at night for about three months by a closely fitting bandage or skull cap.

COMMENTS

In our experience, we have never seen any of the maneuvers practiced by anxious mothers in trying to train prominent ears back into normal position accomplish the desired result.

Except in the mildest cases, excision of the skin alone with closure will not be permanently effective, since the spring of the cartilaginous framework of the ear is so continuously persistent that in time it will overcome any soft part closure; the scar and adjacent tissues will stretch and the deformity will recur. In order to obtain a permanent result, it is essential to break the spring of the cartilage of the ear.

By the removal of long elliptical or crescentic strips of cartilage with turning in of the edges or by properly placed incisions through the cartilage, the antihelix and other supporting cartilage ridges can be reconstructed. In some cases, it is necessary to excise more than one piece of cartilage or to break the spring of the cartilage in more than one place according to the degree and location of the deformity.

It is not possible to give specific directions as to exact amount of cartilage and skin to be removed, since each case varies and each ear in each case presents a different problem which must be solved individually.

Care must be taken to replace the two ears fairly symmetrically. If the denudation of skin behind the ear is too wide, we will find the retroauricular angle almost obliterated. The ears should not be made to adhere too closely to the skull, since an ear plastered permanently against the skull is almost as objectionable as one that is too prominent.

When an ear is abnormally large, in addition to being unduly prominent, the appropriate plastic operation which will reduce the size as much as is desired should be done, in addition to restoring the ear to its normal position.

After removal of portions of cartilage and reconstruction of the antihelix, the skin on the anterior surface may occasionally be thrown up into an abnormally prominent fold. This fold will often smooth itself

down in time, but if it persists it can be reduced in size without difficulty at a secondary operation. One ear may be replaced at a time, but our preference is to operate on both under the same anesthesia.

The brilliant green puncture marks in the skin of the ear disappear within a few days and are frequently gone by the time the final dressings are removed. Unless something is done for these patients to correct the deformity, frequently we find that there is serious interference with complete mental and physical development.

Ordinarily we do not operate on children with abnormally prominent ears until they are four or five years old. We have operated on adults as old as forty-five years of age. As far as we can ascertain from observation of cases operated on when they were children, the operation described causes no interference with the normal growth of the ear.

SUMMARY

The use of the brain table in allowing exposure and comparison of both ears at the same time in the desired position is advantageous. The object of the procedure is to reconstruct the undeveloped or unfolded portion of the antihelix so that a rigid buttressing ridge, or ridges, will be formed which will support the ear in its normal position.

Perforating the tissues with the needle dipped in brilliant green solution in marking out the line of cartilage to be incised, or excised, is a distinct advance in technique. The importance of completely breaking the cartilage spring by incision or excision is emphasized, as we have never seen a permanent result unless the spring of the cartilage was broken.

In the method described, the sutures used in turning forward the cartilage edges to form the antihelix are all placed in the perichondrium, as the cartilage itself is friable, and none are placed in the periosteum of the skull.

The use of the seasponge as a postoperative dressing has been very helpful in fixing the reconstructed ridges and in immobilizing the entire ear.

After successfully restoring prominent ears to normal position, the appearance of the patient is vastly improved; the mental aspect is entirely changed for the better; and many adults are able to obtain suitable employment from which they had been barred on account of their bizarre appearance.

We have been able to follow some of our patients for years and have found that the procedure we have outlined is simple and satisfactory and that the results are permanent. So looking at the deformity both from the psychologic and the practical standpoints, we feel that the restoration of prominent ears to normal position is without doubt justified and advisable.

A HISTOLOGIC STUDY OF THE INTESTINE PROXIMAL TO CARCINOMA OF THE RIGHT SIDE OF THE COLON, ASSOCIATED WITH ANEMIA

C. Stuart Welch, M.D., Fellow in Surgery, the Mayo Foundation, Charles W. Mayo, M.D., Division of Surgery, and E. G. Wakefield, M.D., Division of Medicine, the Mayo Clinic, Rochester, Minn.

IT SEEMS to be generally believed that severe anemia associated with carcinoma of the cecum and the right half of the colon is due to some mechanism other than loss of blood from the ulcerated tumor.[3, 5] This impression has been general and often may be applied to distinguish clinically the symptoms of lesions in the cecum and ascending segment of the colon from those produced by lesions in the distal portions of the large intestine. That is, in carcinoma of the cecum the associated anemia may be severe and occasionally the only symptom. The anemia present in carcinoma of the right half of the colon is usually of the hypochromic type, which may be attributable to hemorrhage. However, cases are occasionally observed in which there is a macrocytic type of anemia similar to pernicious anemia. The blood findings do not therefore seem to have any direct relation to the presence or the absence of macroscopic blood in the feces. These circumstances form the basis for the opinion that carcinoma of the right half of the colon may exert a special influence on the blood findings in this disease.

Recent work, which was concerned with the relation of the gastrointestinal tract to the formation of blood, has demonstrated certain possible relations between the anemia and the physiologic disturbances produced by the lesions in the intestine. These physiologic disturbances may result from a number of pathologic conditions of the bowel;[2, 4, 7] in these conditions it has been suspected that anemia resulted from failure of the intestine to absorb iron and the hematopoietic principle. That is, there may be direct loss of hematopoietic substance or iron, such as could occur in the case of multiple intestinal anastomosis, diarrhea, and fistula. There also may be an alteration in the intestinal mucosa which results in malabsorption of antianemic substances; into this category should fall those benign and malignant lesions of the bowel which are associated with rapid elimination of intestinal contents.

The question of etiology of the anemias in intestinal diseases without gross hemorrhage has been approached experimentally by Seyderhelm, Lehmann, and Wichels.[6] They studied the blood of dogs which had intestinal strictures. Of ten dogs that had satisfactory intestinal stric-

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tures, a macrocytic type of anemia occurred in two. In these dogs there was marked infection of the bowel above the site of the stricture. These authors therefore attached considerable importance to structural alterations caused by infection in the intestine proximal to the disease accounting for the anemia. The object of this work is to investigate the relation of such experimental findings to the explanation of the anemias which occur in carcinoma of the right colon of man. That is, an attempt was made to determine whether or not there were changes in the anatomic structure as the result of infection or other disease in the intestine proximal to carcinoma of the right half of the colon. Carcinoma of the cecum and ascending colon does not commonly result in marked obstruction unless intussusception occurs, but it may offer some barrier to the fecal current for a relatively long time and is situated at a strategic point for exerting influence on the small intestine.

We used surgical specimens which are most desirable for study since they are fixed immediately after removal and are free from changes in the intestinal mucosa which might be found in postmortem specimens. Ten cases of carcinoma of the right half of the colon, which was associated with anemia of varying severity, were studied. A description of these lesions and certain clinical data relative to the blood are recorded in Table I. Microscopic studies were made of the walls of the large intestinal proximal and distal to the growths. Similar studies were also made of the ileum near the region of the ileocecal valve and at variable distances proximal to this region, depending on the amount of ileum removed at operation. Sections were stained with hematoxylin and eosin.

TABLE I

SITE OF THE LESIONS AND BLOOD FINDINGS IN CARCINOMA OF THE RIGHT SIDE OF THE COLON

CASE	SITUATION OF CARCINOMA	ERYTHROCYTES (PER CU. MM. OF BLOOD)	HEMOGLOBIN (GM. PER 100 C.C. OF BLOOD)
1	Ascending colon	4,090,000	10.7
2	Cecum and ascending colon	4,340,000	9.5
3	Cecum and ileocecal region	4,320,000	7.3
4	Hepatic flexure	3,290,000	10.7
5	Ascending colon	4,140,000	10.1
6	Ascending colon	3,800,000	8.5
7	Ascending colon	3,450,000	9.5
8	Ascending colon	3,970,000	8.5
9	Ascending colon	2,600,000	7.0
10	Hepatic flexure	4,140,000	11.7

No significant and constant changes were observed in the mucosa, submucosa, or muscular coats of the large intestine. The colon proximal to the growth was not different from that distal to the growth. Infiltration with lymphocytes and polymorphonuclear leucocytes was not prominent and never marked in the sections of the bowel taken from

regions above the lesions. Histologic examination of the ileum showed that the intestinal epithelium was not altered; the villi were normal and submucosa did not show any change. In none of the specimens examined were there changes in the intestinal wall which might suggest the possibility of alteration in the functional activity of the bowel.

COMMENT

It is important to know that the segments of the terminal portion of the ileum which have been studied showed no important changes in structure, although the patients had moderately severe anemia. There was no evidence of atrophy of the mucosa nor were there infiltrative changes in the bowel wall. The possibility of an absorption defect for antianemic substances in this condition seems to us less likely in explaining the anemia than the probability of loss of blood as a result of hemorrhage from the neoplasm. Basing their explanation on rational circumstantial evidence, Alvarez and his associates[1] have accounted for these anemias on the basis of bleeding. Carcinomas of the right half of the colon are usually larger and of longer duration than are those in other portions of the large intestine. About 15 per cent of carcinomas of the right half of the colon are symptomless, except for anemia which develops slowly. These patients may suffer a slow but constant bleeding, which is not macroscopically detectable in the feces.

Whatever the cause of anemia in cases of carcinoma of the right half of the colon may be, we do not believe it is due to objective structural changes proximal to the lesion in the colon or in the terminal portion of the ileum.

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COMPLETE COSTECTOMY

A New and Simple Method of Rib Excision Applicable in Thoracoplasty and Chronic Empyema*

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THE gain for the patient and the risk assumed are the chief considerations in deciding upon the advisability of an operation of election. The risk of an operative procedure is difficult to gauge in a given instance. No experienced surgeon essays to do it with absolute accuracy. Unpredictable findings and occurrences in an operation still attach something of the uncertainty of prophecy to the estimate of the surgical risk. Apart from the risks peculiar to operations upon certain tissues or organs, the principal concern relates to the formidableness of the procedure and the physical status of the patient. Not uncommonly, patients whose ability to withstand surgical intervention is poor need operations of the greatest magnitude. No wonder that the surgeon's responsibility is great and that he occasionally shortens life when he proposes to lengthen it or make it happier.

Thoracoplasties for tuberculosis or extensive chronic empyema as currently performed are formidable operative procedures; and the patient, who with the aid of his surgeon's counsel must weigh the gain and risk involved, is often in poor physical condition. The innovation of alternative remedial measures or operations of lesser magnitude which will accomplish the same end are urgently needed in order that the hazard may be lessened and the benefits of such therapeutic agencies be made available to a larger group of despairing patients.

The operation of thoracoplasty for pulmonary tuberculosis consists of a series of one to four or more operations in which the paravertebral portions of the ribs are exposed through long incisions. Because of the difficulty of exposure, the incision for the removal of the upper ribs is of necessity greater in length than the segments of rib removed. The general plan of complete or subtotal removal of the upper three ribs with excision of lesser lengths of succeeding ribs in multiple stages is now the generally accepted plan of operation. The present trend has been toward a more adequate collapse of the upper lung by more extensive removal of the first to sixth or eighth ribs preserving the lower bony thorax intact.

The general principle of an incision approximately as long as the rib to be removed is described in all texts upon thoracic surgery. That

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extensive incisions through large muscles, such as are to be found on the posterior aspect of the thorax, invite considerable blood loss and are potentially shock producing every surgeon of experience knows. Gatch and Little have determined the amount of blood loss in operations of various kinds and have indicated that incisions through large muscles are usually attended by greater loss of blood than those practiced in meticulous hemostasis would anticipate.

Alexander has sketched the early history of thoracoplasty and has summarized the views concerning the technique of its performance which serve as the basis for the modern operation.* The general plan of procedure in extensive chronic empyema is essentially the same, longer segments of the lower ribs, however, being excised and sacrifice of the thickened parietal pleura often being mandatory to obliterate the empyema cavity.

THE AUTHOR'S OPERATION OF RIB REMOVAL

Every surgeon who strips a rib from its periosteal bed, preparatory to its removal, takes advantage of the direction of insertion of the external intercostal muscle bundles upon the rib. On the upper side, he begins behind and strips in a forward direction with an appropriate instrument; on the lower margin this rib is freed by stripping in the opposite direction. Some years ago, it occurred to the writer that preliminary excisions of the anterior costal cartilages and division of the upper three ribs would facilitate performance of the upper thoracoplasty. My associate, Herbert A. Carlson, made a preliminary report upon this method in 1934 and together with W. F. Bowers we have reported recently a more extended trial with the procedure.

The removal of ribs through short anterior and posterior incisions would appear to be a natural sequel to that method of rib excision. The writer has had occasion to prove to his own satisfaction that the entire rib can be removed in this manner. First, the rib is divided anteriorly either at the sternum or at the chondrocostal junction and the intercostal muscle bundles are separated along the lower margin of the rib. At a second posterior operation, the rib is divided at the site of articulation with the transverse process, the intercostal muscle bundles are separated from the superior border of the rib, and the rib is then pulled out through the tunnel of the posterior incision. In both the anterior and the posterior operation, the surgeon stands on the contralateral side.

TECHNIQUE

The Anterior Operation.—The patient lies flat upon his back on the operating table. When the costal cartilage and anterior end of the first rib are to be excised, a routine procedure in thoracoplasty for tuberculo-

*In Alexander's new book *The Collapse Therapy of Pulmonary Tuberculosis*, which has appeared since this paper was written, there is an excellent summary of various manners of doing the anterior operations.

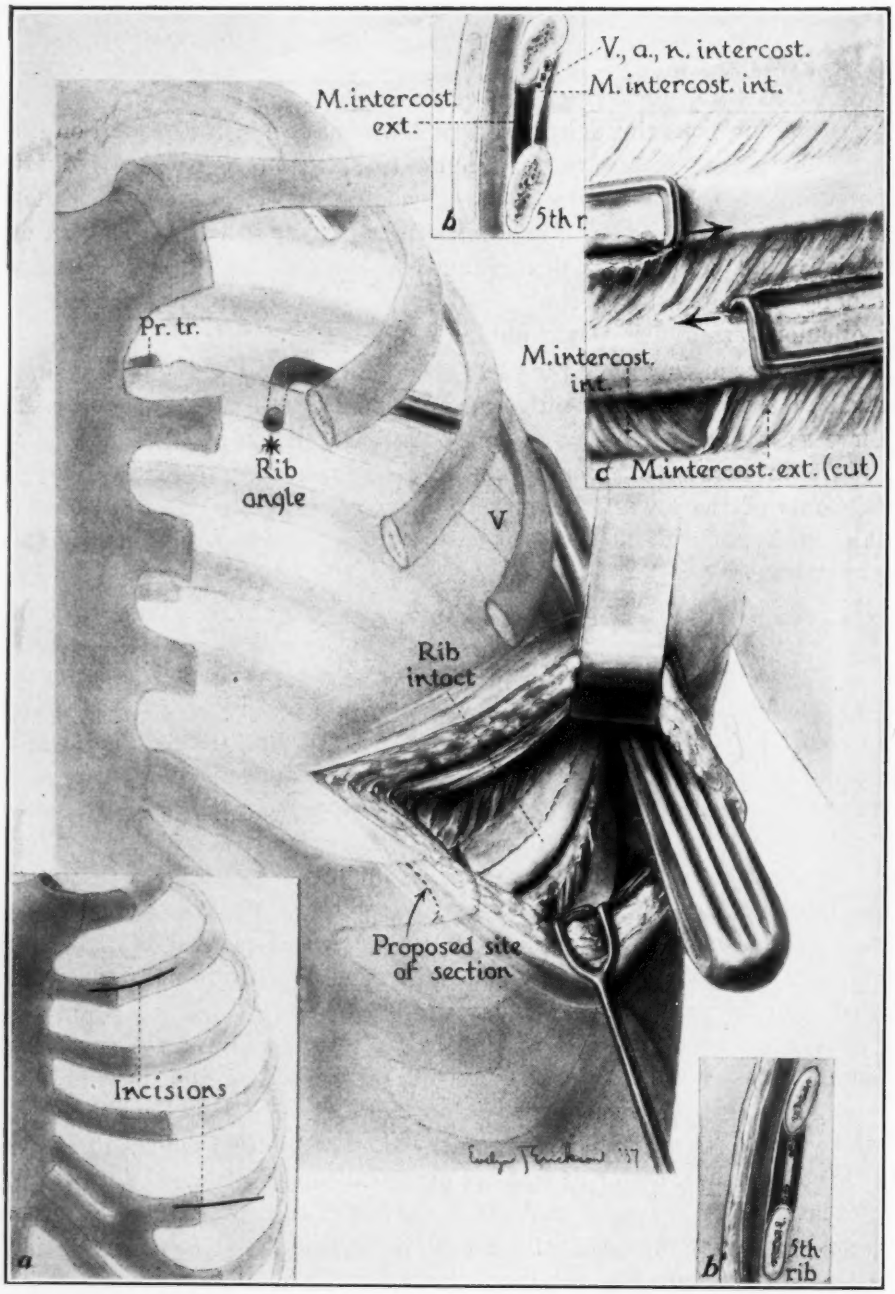

Fig. 1.—The first stage of complete costectomy—the anterior operation. The periosteal stripper is being pushed along the lower margin of the fifth rib. It has not yet been sectioned anteriorly. The extent of bone and cartilage removal (extrapleural thoracoplasty for tuberculosis) of the first to fourth ribs is shown. The stripper can be pushed as far as the angle of the rib posteriorly. The muscles of the lateral thoracic wall are elevated on a retractor in the interspace as the periosteotome is pushed along the lower edge of the rib. *a*, The anterior incisions (see text). *b*, The nature of attachment of the external and internal intercostal muscle fibers to the ribs in the vicinity of the angle of the rib; on the upper rib, shown in this insert, it is apparent that the periosteal stripper pushed posteriorly along the lower margin of the rib will detach only the fibers of the external intercostal muscles. On the lower rib, however, it is evident that the stripper will detach both internal and external intercostal muscle bundles (see text). *b'*, The nature of attachment of external and internal intercostal muscle fibers to the rib ventral to the axillary line. It is apparent that the periosteal stripper will separate both muscles from the rib when pushed along the borders of the rib. *c*, The direction of insertion of the external intercostal muscle bundles accounts for the simplicity of this method of rib removal. The internal intercostal muscle fibers, it is to be observed, run in the reverse direction. The external intercostal muscle bundles are deficient anteriorly between the costal cartilages; the internal intercostal muscle bundles are missing posteriorly, proximal to the angle of the rib.

sis, the arm is extended and abducted upon an arm-board to alter the direction of the subclavian vessels and elevate them from their normal course over the first rib. A short transverse skin incision about five to eight centimeters in length affords ready access (Fig. 1*a*). The fibers of the pectoralis major muscle are separated; the periosteum is incised, and a periosteal separator of the O'Brien type is inserted between the lower margin of the rib and its periosteal bed; with surprising ease, this instrument can be pushed along the edge of the rib as far as its angle posteriorly (Fig. 1). If care is observed in its withdrawal, the hook

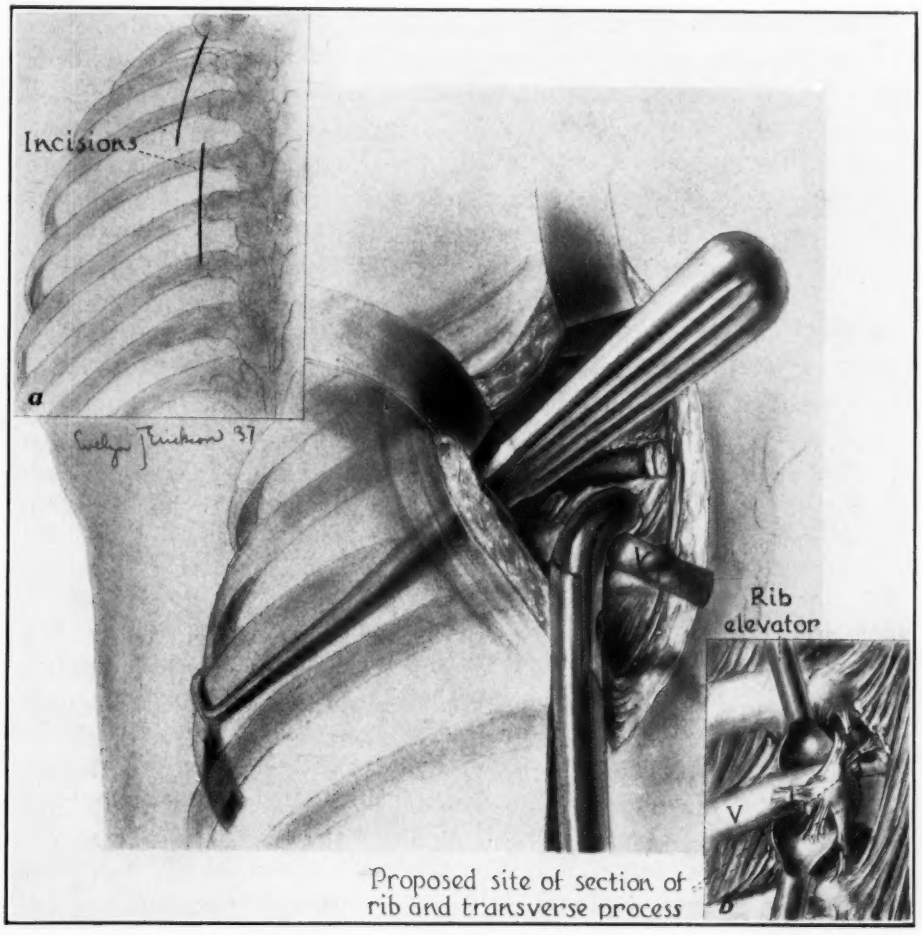

Fig. 2.—The posterior operation in which the ribs are removed. The stripper is being pushed in a forward direction along the superior border of the rib. After exposure of the rib through the incision as shown in Fig. 2*a*, the costotransverse articulation is exposed (Fig. 2*b*) by cutting and separating the short and long levator muscles of the rib as well as the multifidus and intertransverse muscles and the intertransverse and costotransverse ligaments. After all the ribs to be removed have been sectioned, the rib is rotated into the wound and its separation from its periosteal bed proceeds as shown. By gentle pushing with a small sponge on a long curved hemostat, the residual attachment of internal intercostal muscle fibers (see Fig. 1 and legend) and that of such accessory muscles as the pectoralis minor or serratus anterior can be dealt with quite easily (see text). Elevation of the muscles of the chest wall on a retractor in the intercostal space is a valuable expedient which facilitates this procedure.

will not engage in the tissues of the chest wall. The rib is then sectioned anteriorly at the point selected for its division, and the overlying muscles of the chest wall are elevated on a Deaver type of retractor with a long but narrow blade. The rib is now seized and elevated with a lion-jaw forceps. With a gentle push of a sponge upon a curved hysterectomy clamp, any fibers of the external intercostal muscle still attached to the lower margin of the rib are readily separated. About 3 to 5 cm. of the anterior end of the rib is then excised. By sliding the incision up or down with appropriate retraction, three or four ribs can be dealt with in the manner here described. Bleeding is minimal. A few ligatures of fine silk suffice for secure hemostasis and the separated fibers of the pectoralis major muscle are approximated by a few interrupted sutures. An external compression bandage as described elsewhere[7] is applied.

The reaction following this procedure is usually slight and in from seven to ten days the ribs can be divided posteriorly and removed. In the anterior operation for tuberculosis, I have removed regularly the costal cartilages of the upper three ribs and since May of this year, when the operation here described was first done, on several occasions I have removed the cartilages from the upper seven ribs. The degree of collapse obtained by this type of procedure has been very satisfactory. Bone not cartilage regenerates from the perichondrium and an infirm anterior chest wall has not been observed following excision of the costal cartilages. Roentgen films following thoracoplasty employing this method of operation suggest that an earlier and more liberal calcification occurs along the periosteal beds than with the usual thoracoplastic procedure.

THE POSTERIOR OPERATION

In the excision of the upper ribs which lie beneath the scapula, the patient is placed prone on the operating table with the neck flexed and the face engaged in the type of head rest employed for operations upon the cerebellum. The lucid drawings made for me by Miss Evelyn T. Erickson, of the Department of Medical Art of the University of Minnesota, illustrate well the technical details of both the anterior and the posterior operations.

An oblique incision indicated in Fig. 2*a*, as employed by White, Smithwick, Allen and Mixter, is probably to be preferred in the excision of the upper three ribs. The fibers of the trapezius and both rhomboid muscles are split and those of the erector spinal group (iliocostalis and longissimus) are separated and with appropriate retraction direct access is obtained upon the neck of the rib and the transverse processes of the vertebrae. The intertransverse muscles and ligaments and the multifidus muscles and posterior costotransverse ligaments overlying the costotransverse articulations are snipped with scissors and separated with a suitable dissector until the neck of the rib and

the distal half of the transverse process are well exposed (Fig. 2*b*). Periosteal separators of the Hedblom type are then placed beneath the rib and transverse process and both are cut through with a large costotome (Horsley laminectomy or Bethune rib shears). When the ribs to be removed have all been cut, each in turn is seized with a lion-jaw forceps. The scapula is not seen during any stage of the posterior operation and there is no necessity to retract it. The rib is elevated and rotated into the wound. Forcible elevation of the muscles of the chest wall upon a retractor with a long but narrow blade inserted in the interspace overlying the rib to be removed is an important item in the technique, as it is in the anterior operation. A periosteal separator of the O'Brien type is now pushed along the upper margin of the rib, freeing it from the external and internal intercostal muscle bundles. The fibers of the external intercostal muscle bundle proximal to the angle of the rib on its lower margin are separated by pulling the periosteotome posteriorly. The periosteal separator is then pushed forward along the lower margin of the rib and apart from the attachment of some of the fibers of the internal intercostal muscle bundle and those of such muscles as the pectoralis minor or serratus anterior, the rib is wholly free. By plying the periosteal separator alternately along the outer and inner surfaces as well as along the upper and lower margins over the entire length of the rib, aided by gentle pushes with a sponge mounted on a long forceps, the whole rib is usually removed with ease. When the anterior scalene muscle has been detached from the scalene tubercle at the anterior operation, the first rib can also be removed in this same manner. The wound is closed with a few sutures of buried silk. In pushing the attached muscle fibers off the rib with a sponge, there is occasionally some bleeding—probably from an intercostal vein. The wounds are invariably dry, however, when closed, and no drainage is employed; and without exception primary healing has been obtained in all thoracoplastic wounds (sixteen patients). More detailed observations will be narrated in a subsequent report.

Twice, a small rent has been made in the pleura (anterior operation) which occasioned no difficulty. I have satisfied myself by dissections on the fresh cadaver at postmortem examination that a rib can be stripped in either direction by this method in the presence of a normal pleura without tearing it. A small abnormal bony excrescence on the rib at the site of muscle insertion is the usual cause of this accident. With the help of Mr. John Phelan, of the Scientific Apparatus Shop of the Department of Physics at the University of Minnesota, I hope to develop improved instruments which will facilitate this procedure. In general, the O'Brien rib stripper is quite satisfactory for the anterior operation. The hook is probably somewhat longer than it need be and the shank should be a little longer. It is interesting to speculate on the development of an instrument which will strip the rib, then cut it and

permit separation of the periosteum on the opposite side as the instrument is withdrawn. Whereas the construction of such an instrument for use in chronic empyema is entirely feasible, it does not appear likely that in the presence of a normal pleura its employment would be as safe as a two-stage procedure.

THE REMOVED RIBS

A few short strands of muscle fibers often remain attached to the removed ribs in the presence of a normal pleura (thoracoplasty for tuberculosis). I have previously[6] directed attention to the ease with which ribs may be separated from their periosteal beds in the presence of chronic empyema and suggested at the meeting of the American Thoracic Society in 1935 that long lengths of ribs could be removed by a process of tunneling in chronic empyema. As may be supposed, therefore, the very longest ribs can be excised in chronic empyema with this method, and the removed ribs often fail to exhibit traces of attached muscle fibers. Coincident with thickening of the parietal pleura in chronic suppuration of the pleural space, changes (enlargement) in the rib occur, in consequence of which the envelopment of the rib by the periosteal sheath becomes much looser.

THIS METHOD OF RIB REMOVAL IN CHRONIC EMPYEMA

Two total empyemas in which expansion of the lung could not be effected have been satisfactorily collapsed with rib removal alone without removal of thickened parietal pleura. One of them concerned a young woman in extremely poor physical status with a tuberculous empyema. The parietal pleura in areas was 2 cm. in thickness. Yet this large cavity was collapsed by four relatively simple operations. The second to the tenth ribs inclusive were divided anteriorly in two sittings through two transverse incisions. In two subsequent operations employing vertical incisions posteriorly (the lower first), the first through the eleventh ribs were divided and removed. The other patient was a young man with a total empyema and a fistula of the thoracic esophagus which occurred in consequence of pneumonia, pulmonary and mediastinal abscess, nine months previously. Save that a small fistulous tract, whose capacity is about 10 c.c., still leads to the esophageal fistula which is still open, the empyema cavity has been completely obliterated and without excision of parietal pleura.

Two near total empyemas have also been completely closed with a similar plan of operation. In another patient with a persistent chronic empyema with enduring bronchial fistula who had undergone three thoracoplastic operations for its obliteration elsewhere twenty years ago, I found that this plan of operation was not suitable. I divided the rib anteriorly (second through tenth in one operation) and removed segments up to the regenerated rib, later doing the same posteriorly. It was

my thought that the regenerated rib could be left on the parietal pleura and a satisfactory obliteration of the cavity would be obtained. At the second operation, however, the posterior thoracic wall was found to be so rigid that it was necessary to excise a segment of the parietal pleura to effectually obliterate the cavity, a pedicled muscle slip being simultaneously sutured over the bronchial stoma.*

In the small chronic empyema where the excision of short segments of two to three ribs will satisfactorily obliterate the residual cavity, the plan of operation suggested here will have no place. In the chronic empyema cavity of intermediate size, which has had no previous thoracoplastic repair, the excision of the overlying ribs by the method here described would appear to be a valuable procedure in that the parietal pleura need not be sacrificed. Some time ago,[6] I reported upon a plan of saving the parietal pleura by a process of ribboning in order to prevent the occurrence of abdominal hernia in operations for extensive chronic empyema cavities. The plan of operation described here, however, is from many standpoints a great improvement over that procedure. The pleural space cavities persisting after total pneumonectomy, it would appear, could be obliterated simply and effectively by complete removal of the ribs overlying the cavity by this plan of operation.

REACTIONS ATTENDING THE OPERATION

The lesser operative reaction attending anterior chondrocostectomy preliminary to the performance of the usual posterior thoracoplastic operation has previously been discussed.[7] Rib excision with avoidance of long posterior incisions has also contributed materially to decreasing the operative as well as postoperative reaction. Dyspnea has not been observed in any of the patients with pulmonary tuberculosis for whom thoracoplasty has been done by this method. This occurrence suggests that extensive division of the muscles of the thoracic wall, as necessitated by the usual thoracoplastic operation, may be even a more important factor in the production of mediastinal flutter and dyspnea than is rib excision. Whether a more satisfactory collapse will be obtained by this than by the usual method of operation remains to be seen. Persistent elevation of the shoulder on the operated side—an unfortunate occurrence which not uncommonly attended upper thoracoplasty performed through a long incision, has not been observed to follow this plan of operation.

It is to be noted that in this procedure the cartilaginous junctions of the rib with the sternum anteriorly and with the transverse process posteriorly are destroyed. When the rib regenerates, these ordinary sites

*This patient, whose cough had been very bothersome, had had frequent pulmonary hemorrhages for years with similar bleeding through the external fistulous tract. Gentle continuous suction was applied to the catheter in the empyema cavity before operation and through convalescence; cessation of cough was immediate and complete. The employment of this expedient in the repair of bronchial fistulas where drainage is great would appear to be distinctly worth while.

for movement of the rib become ossified and fixed. There would appear to be, therefore, an additional theoretical consideration in favor of complete costectomy as the operative procedure of choice in thoracoplasty for tuberculosis. For in addition to collapsing the pulmonary cavities, complete costectomy fixes the chest wall so that the normal motion of the ribs in respiration is lost. The external intercostal muscles (muscles of inspiration) and the internal intercostal muscles (expiratory), to be certain, constitute strong muscle masses which collectively have an enormous power. Yet, when the sites of normal rib movement are destroyed, the to-and-fro motion of this portion of the chest wall in respiration must be minimal.

A condition somewhat disturbing to the surgeon in the acceptance of this plan of operation for thoracoplasty for tuberculosis may be the increased number of operations. However, the patient who does not elect the safest procedure is in my experience, unusual. In chronic empyema the second through the tenth ribs anteriorly have been divided on two occasions in one sitting with surprisingly little reaction. A short time later the fifth through the tenth and eleventh ribs were sectioned and removed posteriorly by this plan of operation in one sitting. In the presence of a thin pleura, as is to be found usually in tuberculosis, I have continued to be conservative with the extent of rib excision in the vertical direction, dividing only three ribs at each operation. In two instances (upper stage) four have been divided and later removed through the posterior incision without untoward effect. The operations can follow one another, however, in five to seven days (ten to fourteen intervening usually between the second and third operations) and the patients leave the hospital just as soon and in much better condition than after the more formidable usual thoracoplasty which taxes the patient's strength and endurance far more. The interval of time between the first and last operations is usually less than a month.

Whereas this plan of operation may extend somewhat the indications for the performance of thoracoplasty to the patient whose physical status is poor, I would utter a word of warning concerning enlarging the indications of thoracoplasty for tuberculous lesions. The operation must not be done on patients with exudative tuberculosis but only when evidence of reactive proliferation and evidence of healing have become manifest.

SUMMARY

A new method of rib removal is described in which the whole rib, or whatever portion is deemed desirable, can be excised through short incisions. Extensive rib excision by this method is less formidable than the usual thoracoplastic operation. In the closure of large chronic empyema cavities, it is particularly valuable in that the cavity may be completely obliterated by relatively simple operative procedures and

without the excision of parietal pleura. The technique of the method owes its simplicity to freeing the rib in the direction of insertion of the external intercostal muscle bundles.

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THE CYTOLOGIC RESPONSE OF THE PERITONEAL FLUID TO CERTAIN SUBSTANCES*

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RECENT advances in the prevention of peritonitis by the preoperative injection of certain substances into the peritoneal space have stimulated considerable research in recent years on the cytologic reactions of the peritoneum to various stimulating agents. It has been established both clinically and experimentally that the introduction of certain substances into the peritoneum will incite the production of a defense exudate consisting of varying amounts of serum, leucocytes, fibrin, and other products, effecting thereby a defense reaction.

This cytologic and physicochemical reaction by the peritoneum to parenterally introduced agents is said to immunize it against infection. Two agents are now generally employed for this purpose: antiperitonitis vaccine made from killed cultures of colon bacilli and streptococci (Rankin and Bargen, 1929), and a purified concentrated bovine amniotic fluid from which the serum albumins and lipoids have been removed (Johnson, 1927). Immunization is induced by injecting either the vaccine or the amniotic fluid concentrate directly into the peritoneum thirty-six to forty-eight hours prior to surgical intervention. The cellular response to the irritant is believed to be at a maximum at this time, and some authors refer to the reaction as a hyperleucocytosis.

Stimulation of the peritoneum to produce an exudate in the body cavity and to produce immunologic reaction was first accomplished some forty years ago. Issaeff and Ivanoff in 1894 showed that they could protect animals from cholera by previously injecting various substances into the peritoneal space. They used water, urine, bouillon, serum, nucleic acid, and tuberculin, and thereby induced a hyperleucocytosis in the peritoneal exudate sufficient to protect against cholera. Durham, in 1897, protected rabbits against typhoid bacilli by previously injecting sodium chloride, and he recommended that prophylactic intraperitoneal injection of suitable sterile material be made twenty-four hours before opening the peritoneum when there was danger of contamination. Likewise Solieri (1902) protected guinea

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pigs against colon bacilli infections by previous intraperitoneal injection of small amounts of sodium chloride. He described three clinical cases in which sterile salt solutions were injected prior to operation. Miyake (1904), working with von Mikulicz, used preparations of killed colon bacilli as well as materials previously used by Issaeff and Ivanoff. He described marked increases in the numbers of cells in the peritoneal transudates. Miyake protected rabbits from peritonitis of fecal contamination by inducing a hyperleucocytosis by injections of nucleic acid or killed cultures of colon bacilli. On the basis of Miyake's work, von Mikulicz (1904) used nucleic acid and sodium chloride in a large series of clinical cases. Aschner and von Graff in 1911, and von Graff in 1912, concluded, however, that no beneficial results were obtained from using the preparations of Miyake and von Mikulicz.

Following these earlier attempts to immunize the peritoneum, very little progress was made in this direction until the recent decade. Johnson (1927) recommended the use of amniotic fluid as a means of preventing peritonitis. Young and Marks (1934) used amniotic fluid in cases of colonic resection; they described a polymorphonuclear infiltration into the exudate of guinea pigs seven hours after the injection of 10 c.c. of the fluid. Herrmann in 1929 prepared a vaccine of killed streptococci and colon bacilli and protected a large number of rabbits from fecal peritonitis. He attributed this protection to some specific local peritoneal immunity. Morton (1929) protected rabbits against lethal doses of hemolytic streptococci, not only by intraperitoneal injections of streptococcic filtrates, but by injections of dextrose, glucose, and sodium chloride as well. Steinberg and Goldblatt (1927[31] and 1928[10]) reported the survival of a large number of dogs, immunized by injections of living and killed colon bacilli, after the intraperitoneal injection of fecal materials. No cellular data were included. Subsequently, in 1933,[32] they reported the cytologic response in the peritoneum of dogs to injections of colon bacilli suspended in tragacanth. Webb (1931) reported on the cellular response in the peritoneal spaces of white rats after the injection of albumin and of carbon particles. The reaction of the human peritoneum to the vaccine was reported by Bargen and others[2, 4, 13, 28, 33] in 1933 and by Dixon and Rixford in 1934. Clinical results following the use of the vaccine were reported by Dixon and Bargen in 1935. Johnson (1936),[16, 17] and Johnson, Coonse, Hazard, Foisee, and Aufranc (1936) reported their studies of the cytologic response in the peritoneum of dogs which had received intraperitoneal injections of amniotic fluid concentrate, colon vaccine, physiologic saline solution, and merthiolate in physiologic saline solution. The maximal cellular reaction occurred in twelve hours in animals that received amniotic fluid and at twenty-four hours in those that received the vaccine. Feagles and Bain in

1936 reported excellent clinical results from the use of a peptone broth in cases of peritonitis. No experimental data on the use of this broth have been assembled.

In most of the studies on the cellular distribution in peritoneal exudates which appear after intraperitoneal injections of the various substances, conclusions were based on the total number of cells per cubic millimeter and on the relative distribution of these cells. No attempts have been made to determine the quantitative distribution of cells occurring in the peritoneum in response to any of the immunizing agents.

This present report covers our study of the qualitative and the quantitative cytologic response of the peritoneum of white rats to antiperitonitis vaccine, to concentrated amniotic fluid, and to sodium ricinoleate. Sodium ricinoleate has been shown to detoxify large amounts of lethal toxins without destroying their antigenic power. When ricinoleate was added to 100 lethal doses of tetanus toxin, the material was rendered harmless, but its capacity to produce large amounts of protective antibodies was retained (Larson, Evans, and Nelson, 1924, 1925).

Sodium ricinoleate is a derivative of castor oil and, according to Rider[26] (1931), is composed largely of ricinoleic acid glyceride. Methods of purification have been described by Rider,[27] so that a chemically pure sodium ricinoleate is now available. It inactivates diphtheria toxins. It is highly hemolytic when given intravenously and causes some liquefaction necrosis when injected intramuscularly. Some irritation is induced when it is given intraperitoneally, but it is better tolerated by animals when given in this way. A review of the experimental and clinical use of the drug up to 1932 has been made by one of us (Seeley).

Rea and Wangensteen (1934) found sodium ricinoleate most effective in the prevention of intraperitoneal adhesions in rabbits. They also found that many of the histiocytes of the peritoneal exudate were either killed or injured by the drug, showing its destructive effect on living cells as well as on bacteria. Wangensteen in 1936[34, 35] reported the use of 1 per cent sodium ricinoleate on all mucous surfaces in cases of gastric resection for cancer, the purpose being to reduce the hazard of infection. Gillette (1936) reported the use of sodium ricinoleate in a series of 300 cases, and he found it of definite value in cases of advanced peritonitis; he believed it preferable to vaccines and amniotic fluid.

Johnson, Coonse, Hazard, Foisee, and Aufranc (1936), however, found sodium ricinoleate a distinct menace in the presence of postoperative infection. They found it extremely irritating and observed widespread cell destruction in the presence of the drug. Corwin, in 1936, working in this laboratory, has compared the cytologic response to

sodium ricinoleate in the peritoneal space of rabbits with the response induced by antiperitonitis vaccine.

METHODS

Only healthy adult white rats of the Wistar strain, weighing from 180 to 225 gm., were used in this study. Using the technique described by Montgomery (1932), a small capillary glass pipette was forced through the abdominal wall into the peritoneal space. The small amount of fluid which readily entered the pipette was placed on a clean slide and an adequate amount was drawn up into a leucocyte counting pipette for determining the total number of cells per cubic millimeter of fluid. The portion remaining on the slide was then stained with Wright's stain or with May-Grünwald-Giemsa stain for the differential count.

In order to determine the total number of cells present in the peritoneal space, it was necessary to know the amount of fluid exudate which was present. This was done by killing the animal by exsanguination, quickly opening the peritoneum, and carefully taking up on previously balanced filter paper all available free fluid. The increase in the weight of the paper was accepted as the weight of free fluid, and 1 gm. of exudate was considered the equivalent of 1 c.c. Knowing the number of cells per cubic millimeter, the number of cubic millimeters of fluid and the differential distribution, it was easy to compute not only the approximate number of cells in the peritoneal space, but also the number of histiocytes, lymphocytes, and eosinophilic, neutrophilic, and basophilic leucocytes as well. Twenty-three healthy male rats were used to assemble the control data with which the data derived from the rats receiving the vaccine, the amniotic fluid, and the sodium ricinoleate were contrasted (Table I).

Eighty rats were used to determine the cytologic response of the peritoneum to each of the three substances tested: namely, antiperitonitis vaccine, amniotic fluid, and 2 per cent sodium ricinoleate.

TABLE I

CONTROL DATA ON PERITONEAL FLUID OF HEALTHY NORMAL RATS

AMOUNT OF PERITONEAL FLUID, MG. (23 RATS)	NUMBER OF PERITONEAL CELLS (100 RATS)		PERCENTAGE OF DISTRIBUTION OF LEUCOCYTES (100 RATS)
	PER CU. MM.	IN PERITONEUM	
223.0 ± 9.9	101,266 ± 3,391	22,582,318	Mononuclears: 79.77 ± 0.28 Eosinophiles: 14.75 ± 0.93 Basophiles: 5.25 ± 0.35 Neutrophiles: 0.08 ± 0.02

Thus a total of 240 test rats were used in the study. Each animal received 1 c.c. of one of these substances intraperitoneally. Ten animals of each of the three groups were killed at 1, 3, 6, 12, 24, 48, 72, and 168 hours after injection.

We determined for each rat at the time of killing the total amount of fluid present in the peritoneal space, the total number of cells present per cubic millimeter of fluid, the percentage distribution of the various cells comprising the exudate, and the total number of polymorphonuclear and mononuclear leucocytes in the peritoneum. The average of the ten determinations made for each item was accepted as representative of the conditions which existed at each period of observation throughout our study.

OBSERVATIONS

Weight of Fluid Present in the Peritoneal Space.—The average amount of fluid present in the peritoneal space of a healthy adult control male rat, according to our method of recovery, weighed 223 mg. (Table I). The data covering the weights of the fluid in each animal killed at each period during the study have been condensed in Table II. It is clear that the injection of either the amniotic fluid concentrate or the antiperitonitis vaccine caused some considerable increase in the amount of fluid exudate in the body cavity, but the amount in those animals which received the sodium ricinoleate was eight to ten times as large as in the control animals. Even at one hour after injection, the peak of the response to sodium ricinoleate had been reached when the volume of exudate recovered was over seventeen times the amount present before the injection was made. The fluid of those animals receiving the amniotic fluid and the vaccine was clear and colorless, but that of those receiving the ricinoleate was blood tinged. The amount of fluid gradually decreased after the first hour, and in the group receiving injections of amniotic fluid and vaccine the amounts recovered were thereafter only slightly in excess of the normal volumes. In the animals receiving sodium ricinoleate, however, the amounts of fluid were always very much higher than in either of the other two groups; and even after one week an average of 855 mg. of fluid was still present in the peritoneal space. This amount was triple that found either in the animals which had received the vaccine or in those which had received the amniotic fluid.

Total Cells Present Per Cubic Millimeter of Peritoneal Exudate.—Our results on the cellular content per cubic millimeter of peritoneal exudate were as follows (Table II): In a series of 100 healthy normal male rats, we found that the average number of cells in the peritoneum was 101,266 per c.mm. of fluid. One hour after the injections were given, when the volume of exudate was high, the number of cells per cubic millimeter was appreciably less and was proportional to the fluid dilution. When the volume of exudate was highest, as in the case of animals receiving sodium ricinoleate, the number of cells per unit volume was least (5,500 per c.mm.). At six hours after injection, the number of cells per cubic millimeter in rats receiving

TABLE II

DATA ON WEIGHT OF PERITONEAL FLUID AND ITS CELLULAR CONTENTS

CONTROLS		MATERIAL INJECTED (1.0 c.c.)	NUMBER OF ANIMALS	TEST ANIMALS							
				HOURS AFTER INJECTION							
	NUMBER OF ANIMALS			1	3	6	12	24	48	72	168
Weight of Peritoneal Fluid (Milligrams)											
223*	23	Amniotic fluid concentrate	10	474	295	255	195	224	220	241	276
		Antiperitonitis vaccine	10	320	282	248	286	377	253	231	288
		Sodium ricinoleate, 2 per cent	10	3,482	3,289	2,574	1,105	1,425	1,417	1,923	855
Total Cells Per Cubic Millimeter of Peritoneal Fluid (Thousands)											
101.26†	100	Amniotic fluid concentrate	10	34.48	97.74	105.41	130.80	116.85	136.10	138.65	140.57
		Antiperitonitis vaccine	10	53.04	93.02	104.95	109.15	121.39	168.10	122.65	123.79
		Sodium ricinoleate, 2 per cent	10	5.50	9.73	12.23	19.85	31.12	36.75	41.34	48.34
Total Cells Present in Peritoneal Fluid (Millions)											
22.58‡	23	Amniotic fluid concentrate	10	16.34	28.83	26.88	25.51	26.17	29.94	33.41	38.80
		Antiperitonitis vaccine	10	16.97	26.23	26.03	31.22	45.76	42.53	28.33	35.65
		Sodium ricinoleate, 2 per cent	10	19.13	31.99	31.48	31.93	44.35	52.07	79.51	41.33

*Weight of peritoneal fluid, milligrams.
†Number of cells per cubic millimeter.
‡Total cells in peritoneum.

amniotic fluid or antiperitonitis vaccine was again at control levels and the amount of fluid more closely approached the normal amounts. In contrast, the peritoneal exudate of rats receiving sodium ricinoleate contained but 12,230 cells per c.mm. at six hours following the injection, and even after one week's time the number of cells per cubic millimeter was still less than half the normal figure.

It is obvious, of course, that tabulations of cells per unit volume of fluid do not reveal the extent of cytologic changes that may occur. When dilution factors are high, then the number of cells counted per cubic millimeter is correspondingly less. Accordingly we have computed the total number of cells present in the peritoneal space of each animal and have thus derived a more adequate basis for comparison of the cytologic response incited by these three irritants.

Total Number of Cells Present in the Entire Peritoneal Space.—By multiplying the number of cells per cubic millimeter of fluid by the number of cubic millimeters of fluid in the peritoneal space, we derived an approximation of the total number of cells in the entire space (Table II). By multiplying our average number of cells per cubic millimeter of fluid in 100 control animals by the average total volume of fluid of twenty-one control animals, we derived a figure of 22,580,000, which we have accepted as the approximate number of cells present in the peritoneal space of each of our normal rats before injection. One hour after injection this number was found to be considerably less. Accordingly, during the hour following injection, millions of cells must have left the peritoneum, to enter either adjacent tissues or the blood stream. Counts taken on the peripheral blood at this time, however, did not indicate that there was any real significant increase in the concentration of leucocytes. At the third hour, however, a real cellular increase had occurred. This was true for all three groups, but especially so for the animals which had received sodium ricinoleate in which case an average infiltration of more than 12,000,000 cells into the peritoneum had occurred in the preceding two-hour period. This marked increase in the cellular content was maintained, although it fluctuated considerably, for the entire week, and even on the seventh day the number of cells in the body cavity of rats which had received the amniotic fluid was still increasing. Those rats which had received the antiperitonitis vaccine showed the maximal total number of exudative cells at twenty-four hours after the injection, but even at the end of the week the total number was still greatly in excess of normal values. The greatest stimulation to cellular proliferation was found in those animals which had received sodium ricinoleate. On the third day, the average of the counts made on ten rats showed a total cell content of nearly 80,000,000. This is nearly triple that found in either the group receiving amniotic fluid or that receiving vaccine, and it is

nearly four times the number encountered in normal animals. Such a cellular response indicates the excessive stimulation which was induced by this irritant.

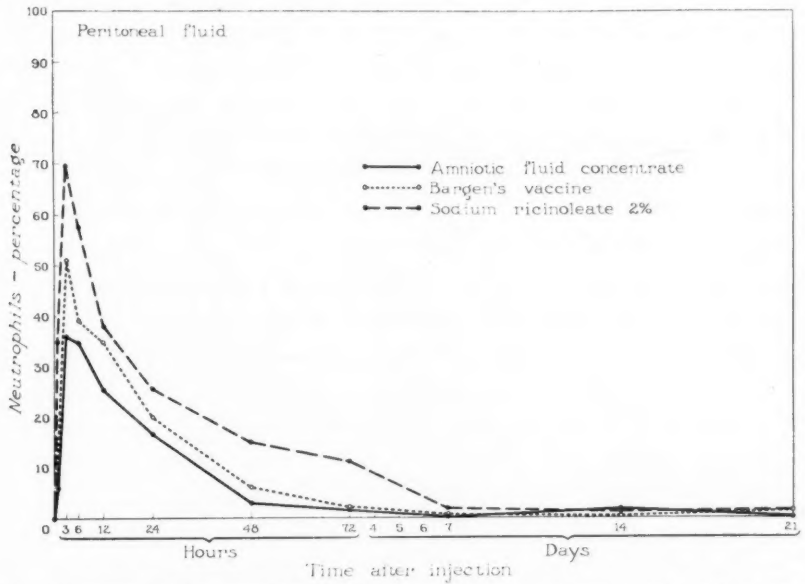

Fig. 1.—Changes in the percentage of neutrophiles in the peritoneal fluid of rats at the different intervals following injection of the three substances.

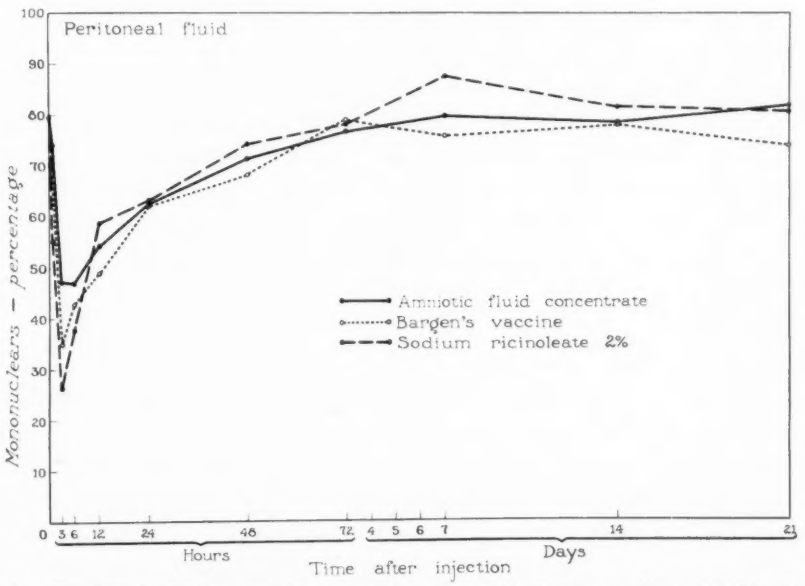

Fig. 2.—Changes in the percentage of mononuclear cells in the peritoneal fluid of rats at the different intervals following injection of the three substances.

The Percentage Distribution of Cells in the Peritoneum.—Although we have computed the complete percentage distribution of the cells in the peritoneal space of all animals killed, we submit in this report the

data pertaining to the percentage distribution of only the mononuclear and polymorphonuclear leucocytes. Basophilic and eosinophilic leucocytes were always present, but their percentages were essentially unaffected by these substances. We have not separated lymphocytes from other mononuclear cells, but we have grouped the histiocytes or clasmatocytes of the exudate with the lymphocytes and have called them "mononuclear" leucocytes. On the basis of 100 counts on 100 animals, we concluded that the distribution of cells in our series of rats before injection was as follows: mononuclear leucocytes, 79.77 per cent; polymorphonuclear leucocytes, 0.08 per cent; basophiles, 5.25 per cent; and eosinophiles, 14.75 per cent.

The data assembled from our tabulation of the percentage distributions of the mononuclear and polymorphonuclear leucocytes in the peritoneal spaces of all animals for the seven days following injection have been condensed into Table III and shown graphically in Figs. 1 and 2. The polymorphonuclear cells appeared in the exudate soon after the injections were made, and, at one hour, percentages of 6.11 and 8.5, respectively, were found for the amniotic fluid and vaccine groups. The neutrophile response to the presence of sodium ricinoleate was most marked. Even at one hour, 35.09 per cent of all cells were polymorphonuclears; at the third hour, 69.65 per cent of all the cells were polymorphonuclears. The neutrophile response to the vaccine was much greater than to amniotic fluid, and in all three groups of animals the highest percentages were reached at three hours following injection. Following this period there was a progressive decline in the percentage of neutrophiles in the exudates, and after one week percentages which were essentially normal were restored. Conversely, the percentage of mononuclear leucocytes progressively increased after the third hour; and on the third day percentages comparable with those of our control group were obtained. Even higher percentages than those were found in the animals receiving amniotic fluid and in those receiving sodium ricinoleate at the end of the week.

Total Mononuclear and Polymorphonuclear Cells Present in the Peritoneal Space.—It is obvious that the percentage distribution will not adequately portray the extent of change which occurred in the exudate relating to the total numbers of mononuclear cells and neutrophiles which were present at the successive intervals following injection. Accordingly, using our data on the total number of cells and percentage distribution, we computed the total numbers of mononuclear cells and neutrophiles which were present in each animal killed at the various intervals following the injection of the three substances. From our control data we had computed that the number of mononuclear cells present prior to injection was more than 18,000,000, and that the number of polymorphonuclear cells was slightly in excess of 18,000. The

TABLE III

DATA ON PERCENTAGE DISTRIBUTION OF AND TOTAL NUMBER OF MONONUCLEAR AND POLYMORPHONUCLEAR LEUCOCYTES IN THE PERITONEAL SPACE

Percentage Distribution

MATERIAL INJECTED (1 C.C.)	NUMBER OF ANIMALS		HOURS AFTER INJECTION							
			1	3	6	12	24	48	72	168
Amniotic fluid concentrate	10	Monos.	74.16	47.21	47.05	54.28	62.88	72.63	77.11	80.07
		Polys.	6.11	36.20	34.23	25.57	16.65	4.32	1.79	0.25
Antiperitonitis vaccine	10	Monos.	68.75	34.95	42.72	48.93	62.82	68.45	79.21	76.21
		Polys.	8.50	51.35	39.49	35.01	20.26	6.45	2.37	0.63
Sodium ricinoleate, 2 per cent	10	Monos.	55.48	26.43	37.82	54.28	63.95	74.52	78.45	87.77
		Polys.	35.09	69.65	57.59	25.57	26.65	15.04	10.95	1.82

Total Number of Neutrophiles (Millions)*

Amniotic fluid concentrate	10		1.00	10.43	9.20	6.52	4.36	1.29	0.60	0.08
Antiperitonitis vaccine	10		1.44	13.47	10.28	10.93	9.27	2.74	0.67	0.22
Sodium ricinoleate, 2 per cent	10		6.71	22.28	18.13	8.33	11.82	7.83	8.71	0.75

Total Number of Mononuclear Cells† (Millions)

Amniotic fluid concentrate	10		12.11	13.61	12.65	13.85	16.45	21.75	25.76	31.07
Antiperitonitis vaccine	10		11.67	9.17	11.12	15.28	28.75	29.11	22.44	27.17
Sodium ricinoleate, 2 per cent	10		10.61	8.45	11.91	12.93	28.36	38.80	62.38	36.28

*Number of neutrophiles in peritoneal cavity of normal rat = 101,266×233×0.08 per cent = 18,065.
†Number of mononuclear cells in peritoneal cavity of normal rat = 101,266×223×79.77 per cent = 18,013,915.

data on these changes (Table III and Figs. 3 and 4) show that, at one hour, more than 6,000,000 polymorphonuclear cells had entered the

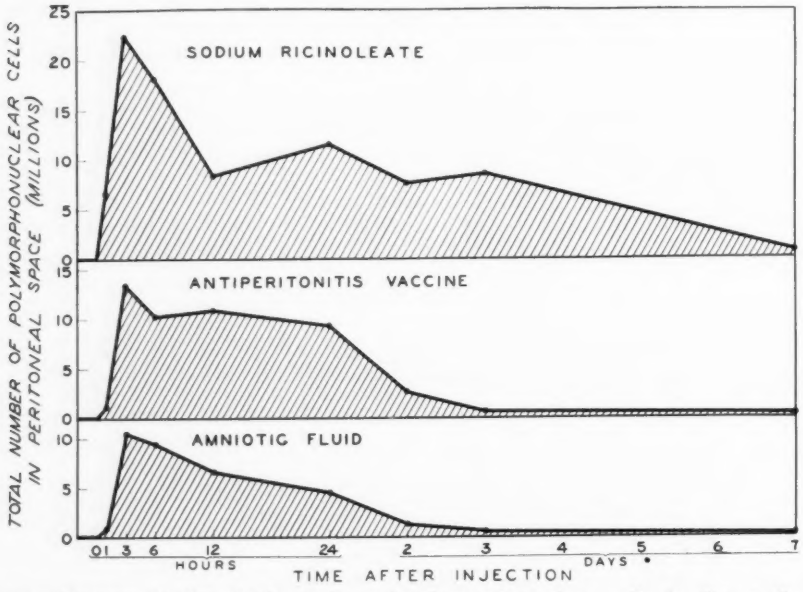
TOTAL NUMBER OF POLYMORPHONUCLEAR CELLS IN PERITONEAL SPACE (MILLIONS)
SODIUM RICINOLEATE
ANTIPERITONITIS VACCINE
AMNIOTIC FLUID
0 1 3 6 12 24 2 3 4 5 6 7
HOURS
DAYS
TIME AFTER INJECTION

Fig. 3.—Changes in the total number of polymorphonuclear cells in the peritoneal fluid of rats at the different intervals following injection of the three substances.

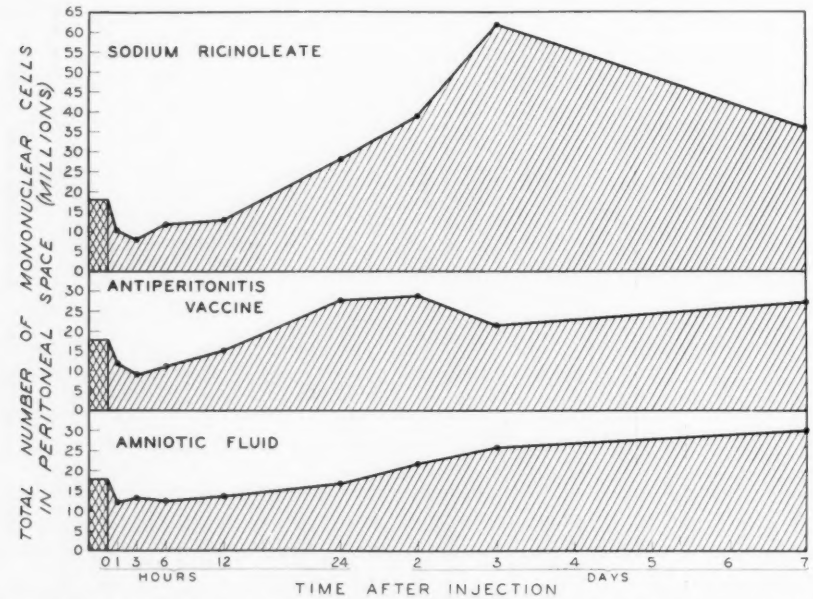
TOTAL NUMBER OF MONONUCLEAR CELLS IN PERITONEAL SPACE (MILLIONS)
SODIUM RICINOLEATE
ANTIPERITONITIS VACCINE
AMNIOTIC FLUID
0 1 3 6 12 24 2 3 4 5 6 7
HOURS
DAYS
TIME AFTER INJECTION

Fig. 4.—Changes in the total number of mononuclear cells in the peritoneal fluid of rats at the different intervals following injection of the three substances.

exudate of those animals receiving sodium ricinoleate and that the number of mononuclear cells was less than when the soap was injected. At the third hour, the infiltration of neutrophiles had reached its height

in all three groups of animals examined, but in those which had received ricinoleate the number of neutrophiles was more than double that found in those receiving amniotic fluid. The rise in the number of mononuclear cells began about the sixth hour after injection. The peak of 62,000,000 cells was found in those animals receiving ricinoleate, and it occurred at the end of the third day. The peak of mononuclear infiltration in animals receiving the vaccine occurred on the second day; whereas, at the end of the week the trend in the absolute numbers of mononuclears was still upward in those animals which had received amniotic fluid.

COMMENT

In this study we have attempted to quantitate some of the changes which occur in the peritoneal cavity of rats after the intraperitoneal injection of 1 c.c. of concentrated amniotic fluid, antiperitonitis vaccine, or of 2 per cent sodium ricinoleate. We have determined the total amount of fluid present, and the percentage distribution and total number of exudative cells present at varying intervals after injection of these three agents.

The results indicate conclusively that all three substances caused a marked increase in the amount of fluid exudate, the increase being much more marked in animals receiving sodium ricinoleate than in those receiving either amniotic fluid or vaccine. Furthermore, the results show that there were in all animals marked influxes of leucocytes into the exudate. In the early period these cells were predominantly polymorphonuclear, but from the twelfth hour onward they were predominantly mononuclear and consisted of monocytes, lymphocytes, and histiocytes. As in the extent of fluid response, the numbers of cells in the exudates of animals which received ricinoleate were usually greatly in excess of those found in animals of the other two groups.

The rate at which these changes in the amount of fluid and in the cellular content occurred, the magnitude of these changes, and the length of time they persisted varied with the material employed.

Conclusions regarding the significance of these results, both as regards the mechanism of response of the peritoneum and the use of these peritoneal irritants in the prevention of peritonitis, must await the results of further investigation. Certain questions which arise concern the life history of these cells which appear in the exudate. Where they come from, how they enter the peritoneum, and how long they remain in the peritoneum are questions which thus far have been answered only in part. Another question is, what is the source of the fluid portion of the exudate? Does it arise from the entire serous surface, or is it mainly elaborated by the parietal or the visceral peritoneum?

While it would be anticipated that a reaction of the peritoneum to a noninfectious irritant would have a protective action against a subsequent infection, as the experimental work of Herrmann, Morton, and others would indicate, it must be admitted that, at present, it is almost impossible to standardize such a process as peritonitis.

We believe that investigations of the sort we are here reporting are of value in filling some of the gaps in our knowledge of peritoneal reactions and of help in supplying the answers to some of the questions enumerated.

SUMMARY AND CONCLUSIONS

This report covers a study of the quantitative and qualitative cellular responses of the normal peritoneums of white rats to three substances: (1) concentrated amniotic fluid, (2) antiperitonitis vaccine, and (3) 2 per cent sodium ricinoleate. One cubic centimeter of each preparation was given intraperitoneally to eighty rats, and ten animals in each group were killed at each of the following intervals after injection: 1 hour, 3 hours, 6 hours, 12 hours, 24 hours, 48 hours, 72 hours, and 1 week.

The following conclusions are indicated:

1. As evidenced by the fluid exudate produced, sodium ricinoleate was found to be far more irritating to the peritoneum than either amniotic fluid or the vaccine. In one hour, eight to ten times as much straw-colored fluid was produced in rats receiving the ricinoleate as in rats of either of the other two groups. Large amounts of fluid persisted even after one week.

2. The total number of cells per cubic millimeter of peritoneal fluid was always less in animals which had received sodium ricinoleate. This is because of the large dilution factor. As between animals receiving amniotic fluid and those receiving vaccine, there were no outstanding differences.

3. The data on the total number of cells in the peritoneal spaces of these animals show that ricinoleate causes an excessive proliferation of histiocytes. On the third day the number of cells in animals receiving ricinoleate was nearly triple that found in animals receiving either amniotic fluid or vaccine. This indicates the enormous stimulative action of sodium ricinoleate.

4. With respect to the percentage distribution of cells in the peritoneal space, the data show that the neutrophile response during the first six hours after injection is much greater in animals receiving ricinoleate than in those receiving either of the other two substances. Likewise, neutrophilic granulocytes remain in the peritoneum very much longer in animals which have received ricinoleate.

5. Correspondingly, the number of neutrophilic granulocytes was always higher, except at the twelfth hour, in those animals receiving ricinoleate than in those of the other two groups.

6. The number of histiocytes in the peritoneal fluid of animals receiving ricinoleate, at seventy-two hours after injection, was nearly triple that found in animals of the other two groups.

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AN EXPERIMENTAL STUDY UPON THE PREVENTION OF ADHESIONS ABOUT REPAIRED NERVES AND TENDONS*

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THE success of many nerve and tendon operations has been prevented by scar tissue which grows about the suture line from the surrounding injured structures. More often than not, the normal muscle plane has been destroyed at the time of injury and only dense fibrous tissue remains. It is this lack of a normal nerve bed or gliding tendon mechanism which creates an important surgical problem.

Many attempts have been made to find a substance which might be used to protect the line of suture, which would form a bed free of adhesions or a smooth gliding surface for a tendon. The variety of materials used clinically and in animal experiments includes fat, cartilage, sterile vaseline, autoplastic fascia, rabbit's vena cava, Cargile membrane, hardened appendices, tubes of magnesium, celloidin, and decalcified bone, gold and silver foil, egg membrane, and formalized arterial sheaths. The results obtained have not been uniformly successful nor have they been duplicated in all instances by other investigators.

J. S. Davis[1] used free fascial transplants with the muscle or inner surface exposed about nerve sutures. Although there was no constriction of the caliber of the nerve after one month, there was very little motion possible within this new sheath. Lewis[2] used fascia and large fat transplants with a fair degree of success about nerve sutures after concluding that fascia alone contracts and becomes adherent. He stated that fat heals in position with but little reaction of the foreign body type and causes the least amount of adhesions. However, Huber[3] found that free autogenous fat transplants were replaced by dense fibrous tissue. Stewart[4] suggested that fat and not fascia should be employed in a neurolysis because fascia produces adhesions which shrink and compress the nerve. In the past year Mayer and Ransohoff[5] have constructed a sheath which resembles the normal tendon sheath lining from tubes of celloidin. They implanted these tubes at the site of the old injury and proposed tendon suture. Six weeks later the proximal tendon stump was threaded through the celloidin tube which was gently removed leaving a smooth glistening

*The materials used in these experiments were furnished by Curity Laboratories of the Lewis Mfg. Co., Walpole, Mass.

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membrane. The tendon was sutured within this new gliding mechanism. Their tubes were made of proxylin and acetone placed in the incubator and allowed to harden over a period of three to five months. More recently, absorbable tubes of catgut membrane have been suggested by Bowen.[6]

Conflicting reports of this character may be found in the literature upon every one of the substances used in an effort to solve this problem. We have confined our study to an experimental investigation of the following materials:

Beef amniotic membrane, which is found in all amniote vertebrat s and which is a purely protective structure in utero, having an inner ectodermal and an outer mesodermal layer.

Fig. 1.—Cross-section of sciatic nerve of dog about which allantoic membrane was placed and allowed to remain in situ 61 days. There is no evidence of the membrane but a thick layer of fibrous tissue, infiltrated with round cells, surrounds the nerve trunk.

Beef allantoic membrane, which is an evagination of the hind gut and which is lined with entoderm and covered with splanchnic mesoderm.

Sheet catgut, derived from the submucosa of the jejunum, stretched, dried, and chemically sterilized.

Beef cecum, chemically sterilized mucosa of the cecum.

Commercial cellophane (cellulose acetate) and *rubber latex.*

Our first problem was to determine the irritative property of each of these structures. Two-centimeter squares of each membrane were placed between the fascia and rectus abdominis muscle of dogs and allowed to remain in situ for various periods of time ranging from 5 to 53 days. The region of implantation was then examined for the signs of inflammation and evidence of absorption. At the end of 5 days, each of the materials was still present but in a mucoid form. Microscopically, the most marked reaction was present in the second

week, but grossly the same mucoid appearance existed with less edema than was present at the end of 5 days. There was little evidence of any one of the materials after the third week and none after the fourth week. The beef cecum was found at the end of the sixth week macroscopically as a minute mucoid patch and microscopically by the presence of leucocytes and lymphocytes. Allantoic membrane was most quickly absorbed and could not be found after the third week.

In a second series of experiments, a plane of cleavage was outlined between the biceps femoris and the semitendinosis muscles in ten dogs. The epimysium of the inner surface of each muscle was denuded by scraping with a scalpel until it was dull and bled slightly. Sheets of allantoic membrane (beef and human), cellophane, and rubber latex were inserted between the muscles, which were then allowed

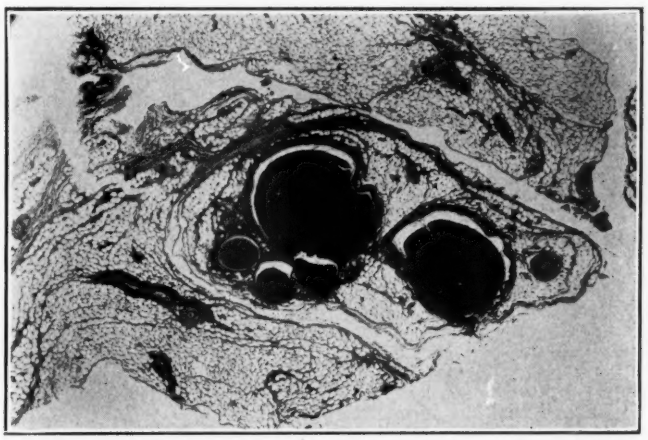

Fig. 2.—Cross-section of sciatic nerve of dog which was covered by allantoic membrane and a pedicle flap of fat and allowed to remain in situ 29 days. The nerve was found lying free in the center of the fat cells without evidence of new fibrous tissue formation and no evidence of the membrane.

to fall together. It was found that after the sixth week allantoic membrane left the most definite plane of cleavage, which allowed the muscles to be separated easily down to the sciatic nerve, while human* and beef amniotic membrane caused a definite obliteration of this plane of cleavage and the muscles were found grown together firmly. Cellophane and rubber latex in thick sheets caused not only severe tissue reaction as indicated by the presence of thick serum in the wound, but there was a definite obliteration of the intermuscular space. In the control experiments, the muscles adhered firmly by dense fibrous tissue.

*Human amniotic membrane was used in these experiments to determine whether or not there was some property of amniotic membrane in utero which prevents adhesions and which may have been destroyed in the preparation or sterilization of the beef membranes. The human amniotic membrane was secured at cesarean section and placed in sterile saline solution. It was then washed and used in all the human amniotic experiments without alteration except where indicated.

A third series of experiments were performed on the sciatic nerves of dogs. Under ether anesthesia the sciatic nerve was exposed in the lower half of the thigh for a distance of two inches. The nerve was roughened with a scalpel until the glossy epineurium was removed and oozing of blood was present. Silk sutures were placed in the epineurium above and below to simulate an anastomosis. Each of the five types of membrane was wrapped about the denuded area of the nerve and the dogs were sacrificed at intervals of from 9 to 82 days. In control experiments the nerve was found firmly adherent to the surrounding tissue, and microscopically a thin layer of fibrous tissue was present about the nerve trunk.

In ten of these experiments, allantoic membrane was used and allowed to remain in situ from 9 to 65 days. Grossly, the nerves were thickened but could be removed from the muscle bed with ease. After

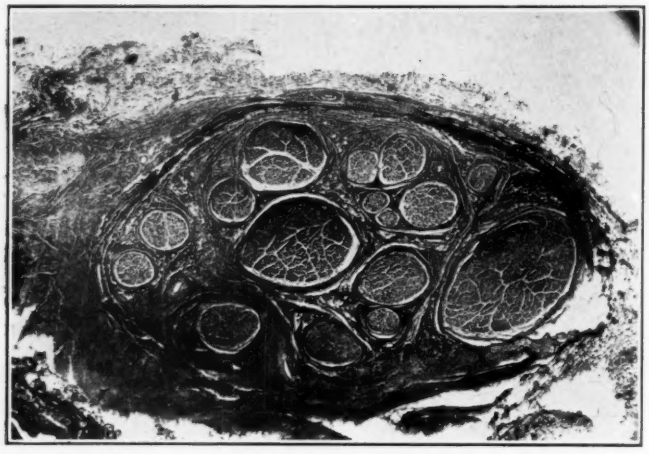

Fig. 3.—Cross-section of sciatic nerve of dog covered with human amniotic membrane, allowed to remain in situ 39 days. There was a definite proliferation of fibrous tissue and marked infiltration of round cells.

the first week the nerve was covered by a loose areolar tissue, while at the end of 7 to 9 weeks, there was no evidence of the membrane grossly, or microscopically. However, a thick layer of fibrous tissue infiltrated with round cells surrounded the nerve trunk so that the nerve fasciculi were constricted. Many small new blood vessels were present in this layer of scar tissue. In one dog, a pedicle of fat was placed about the allantoic membrane. Fine adhesions developed between the fat and muscle, but the nerve was found lying free in the center of the fat cells without evidence of new fibrous tissue formation.

Beef amniotic membrane was used to cover the sciatic nerve of five dogs and allowed to remain in situ from 9 to 65 days. The nerves could not be removed from their beds without injuring the tissue at any time and the immediate covering of the nerve could not be determined grossly. Microscopically, there was a definite proliferation of

fibrous tissue and a marked infiltration with round cells. Fat was also placed about this membrane in one dog and became adherent both to the nerve and surrounding tissue. Human amniotic membrane was used in seven instances and allowed to remain from 39 to 56 days. These nerves were all firmly fixed in position and could not be removed without dissection. Microscopically, there was very little difference between the human and beef amniotic membranes, but if any, the human membrane caused more reaction.

Beef cecum was used about four nerves and allowed to remain in situ from 18 to 26 days, resulting in definite adherence to the surrounding structures. Microscopically, thick dense fibrous tissue with numerous round cells and new blood vessels was present about the nerve trunk.

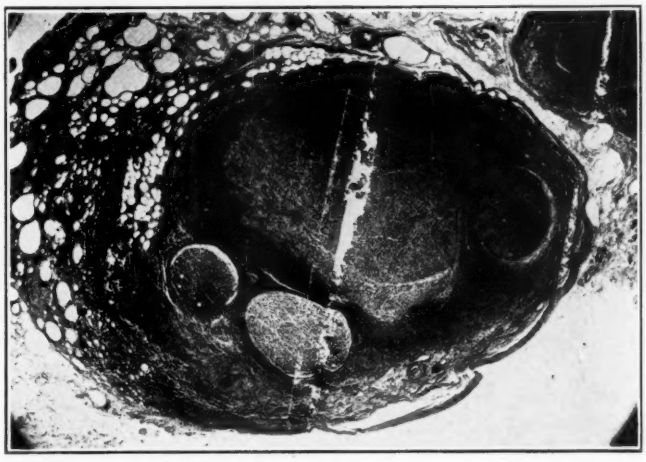

Fig. 4.—Cross-section of sciatic nerve of dog covered with sheet catgut, which was allowed to remain in situ 42 days. There was a thick layer of scar tissue intimately attached to the nerve bundles.

Sheet catgut membrane was used in five dogs and allowed to remain from 13 to 82 days. The nerves could not be removed except by sharp dissection. There was marked thickening of the nerve and its surrounding tissue. Microscopically, there was a thick layer of scar tissue after the third week which was intimately attached to the nerve bundles. Fat placed about the sheet catgut did not prevent the formation of adhesions as was true in the case of the allantoic membrane.

SUMMARY

From the gross and microscopic evidence, it was apparent that allantoic membrane caused the least reaction about the traumatized nerve and protected the nerve from the growth of scar tissue from the surrounding tissue bed. Even better results were obtained with the use of allantoic membrane covered with a flap of fat. Beef and human amniotic membrane produced definite adhesions about the nerve and

fibrous thickening to the same degree as control experiments. Beef cecum and sheet catgut produced thick fibrous adhesions which persisted up to 42 and 82 days and proved to be the least desirable of the membranes studied.

A fourth series of experiments was carried out upon the tendons with and without sheaths in dogs. The Achilles tendon of dogs has no definite sheath but is covered by a paratenon. The peritendinous tissue is important in the reparative process of the tendon in that it provides a blood and lymphatic supply to the injured tendon, and finally after this outer sheath serves its initial function it loosens and takes over a gliding function.[7] In tendons with sheaths, as in the foreleg of the dog, a mesotendon which contains the nutrient blood vessels is attached on the deep surface of the tendon. No matter how carefully one may repair a divided tendon, the endothelial cells lining

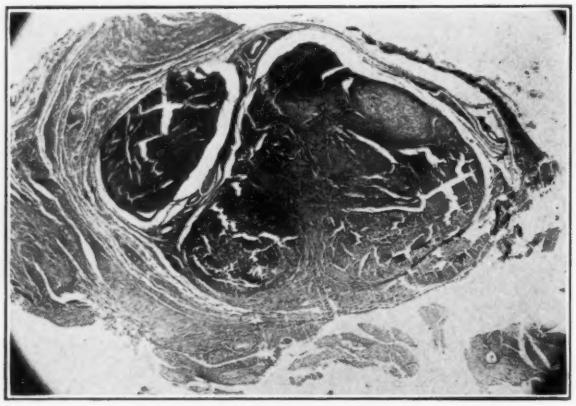

Fig. 5.—Extensor carpi ulnaris tendon of dog. Sheath was opened, tendon cut, sutured, denuded of endothelial surface, and covered with allantoic membrane, which was allowed to remain in situ 37 days. There is no evidence of the membrane, and the sheath of the tendon is not adherent. This compared favorably to the control specimen in which no membrane was used about the tendons with sheaths.

the tendon sheath and surface of the tendon are rubbed off. This predisposes to adhesions and fibroblasts grow out from the subendothelial tissues of the sheath and tendon and unite these structures. However, when the limb is freed and motion begins, the stretching causes an elongation of the connective tissue cells which break and form flat endothelial cells. This process forms a new gliding mechanism.[8]

The following experiments were performed to determine the value of various membranes in forming a sliding mechanism following tendon repair. The Achilles tendon of dogs was exposed by a curved incision on the lateral surface of the leg to prevent adherence of the scar to the tendon. The peritendinous tissue with its vascular plexus was retracted and the endothelial covering of the tendon was removed by scraping the tendon with a scalpel until it was dull and lusterless.

The tendon was then severed transversely and sutured with silk. A thin sheet of each of the various membranes was carefully wrapped about the denuded and sutured tendon and the peritendineum was not sutured but was allowed to fall back over the membrane. Casts were applied to the legs for two to four weeks and the dogs were sacrificed at intervals varying from 9 to 89 days. Comparative studies were also made on the extensor carpi ulnaris and extensor digitorum tendons. The sheaths were opened and the tendon was denuded of its endothelial covering for one inch. The tendon was then sectioned transversely and sutured with black silk. The suture line and denuded tendon were covered with a layer of one of the four types of membrane. The extensor tendon in the opposite foreleg was used as a control in each instance. The tendon sheaths were not sutured but allowed to fall back into place over the sutured tendons. In this

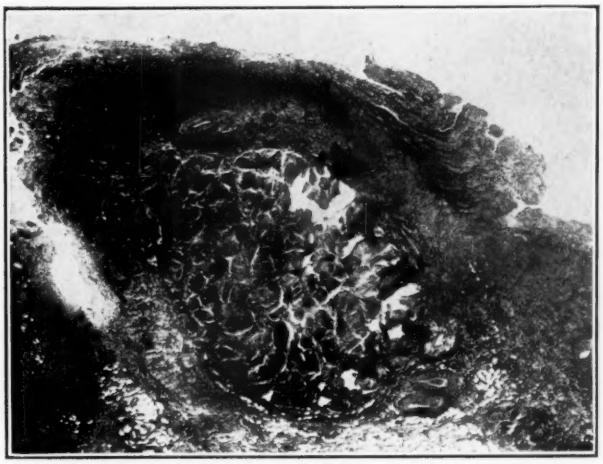

Fig. 6.—Achilles tendon of dog cut, sutured, denuded of endothelial surface, and covered with beef amniotic membrane, which was allowed to remain in situ 15 days. Paratendinous tissue is invaded by numerous leucocytes and the tendon is fragmented.

group of experiments a layer of commercial cellophane (cellulose acetate) was used in several dogs to compare with the animal membranes.

Allantoic membrane was placed about the Achilles tendon of fourteen dogs and allowed to remain in situ from 5 to 82 days. During the first three weeks the tendons were markedly thickened, soft, and presented a dull gray appearance. The peritendinous tissue was thickened to three times its normal diameter. From the fourth to the ninth weeks the tendons became thinner and firmer and the peritendinous sheath was also reduced in thickness. However, at this time the tendon could be moved freely beneath the skin and function was not impaired. On removal of the tendon, it came away from its position beneath the skin easily. The microscopic examination revealed fine attachments of the surrounding tissue to the tendon, espe-

cially about the intratendinous septa even up to the seventh week. In one dog the combination of allantoic membrane covered with a flap of fat was allowed to remain about the Achilles tendon for 82 days. At the end of that time, the tendon could not be separated from the skin due to the dense adhesions present.

Allantoic membrane was inserted about tendons with sheaths and examined after 37 to 89 days. The tendons were adherent to the overlying sheath by easily broken, thin adhesions but moved easily within the sheaths at the end of 89 days. Microscopically, there was an endothelial covering to this tendon and lining of the sheath. This group compares favorably to the control experiments in which no membrane was used about tendons with sheaths.

Beef amniotic membrane was used about eleven Achilles tendons and human amniotic membrane about two. The materials were al-

Fig. 7.—Thirty-seven-day control specimen in which Achilles tendon was sectioned, sutured, and denuded of endothelial covering, but in which no protective membrane was used.

lowed to remain in situ from 5 to 82 days. Grossly, the tendons did not move easily beneath the skin and the surface was dull and roughened. Early the tendons were thickened and gelatinous, while after 49 days there was poor healing of some tendons and the peritendinous tissue was thickened. Human amniotic membrane in larger sheets caused a marked enlargement in the regional lymph nodes and the tendons were fixed to the surrounding structures. Microscopically, the tendon was fragmented and the peritendinous tissue was invaded by numerous leucocytes.

Amniotic membrane was used about five tendons with sheaths and allowed to remain in situ from 37 to 70 days. The tendons moved within the sheath after the sixty-fifth day, but in those tendons where the sheath was destroyed, dense lusterless adhesions were present and motion was limited.

Beef cecum was used about six tendons and allowed to remain in situ from 14 to 37 days. In all these tendons there was marked swelling with poor healing in the first three weeks. The tendons did not separate from the surrounding structures and microscopically there was fibrous tissue present about them.

Catgut in sheets was used about four Achilles tendons and allowed to remain in situ from 27 to 82 days. The late results up to 82 days were similar to those found in control experiments, namely, fixation of the tendon peritendinous tissue and skin about planes of cleavage.

Commercial cellophane was used about tendons with sheaths in 6 dogs. The cellophane was sterilized by immersing in tubing fluid (alcoholic solution of potassium mercuric iodide). There was swelling and fluctuation of the skin over these tendons, and thick serosanguineous fluid escaped upon opening the tendon sheath. The cello-

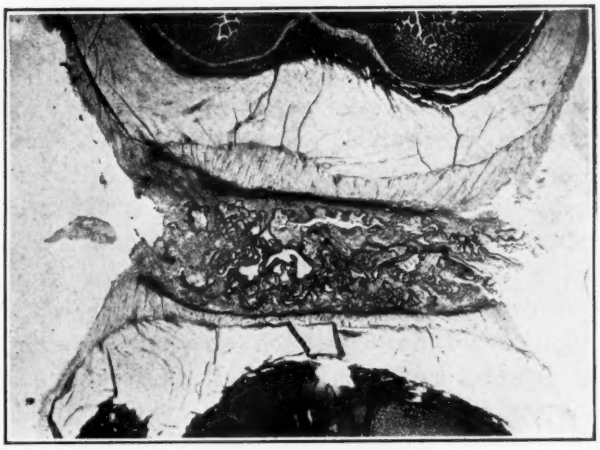

Fig. 8.—Cross-section of two loops of jejunum denuded of their peritoneal surfaces. A layer of beef cecum was inserted between the loops, which were then tacked together and allowed to remain in situ 15 days. Invasion of membrane by small round cells. Ingrowth of scar tissue from subserosa permanently fixed opposing surface of bowel.

phane originally inserted was still present. Three tendons were covered with very thin cellophane and allowed to remain in situ 83 days. There was no reaction within the sheath and the tendon moved easily. The surface of the tendon was well lubricated and presented a shiny appearance. The outer surface of the sheath where it had been elevated from its bed was adherent.

SUMMARY

Achilles tendons sectioned, sutured, and not covered with any membrane heal with definite fixation to their surrounding structures by fibrous adhesions which do not loosen in dogs even as late as the eighth week. Allantoic membrane used over Achilles tendons allows for a peritendinous sheath which is less adherent to the tendon. While not al-

together free of adhesions on microscopic examination, grossly there is free motion of the tendon. The use of beef amnion is not as efficient as allantoic membrane but forms a less adherent gliding mechanism than the control experiments.

Human amniotic membrane, untreated, was definitely inferior to allantoic membrane. The use of beef cecum and catgut in sheets causes dense adhesions, and in some instances the use of catgut causes firmer fixation than that found in the control experiments.

Injured tendons in which the sheath was not destroyed healed with an excellent gliding mechanism after five weeks. The insertion of animal membranes about these tendons forms a gliding mechanism which is inferior to the control experiments in which the sheath is left intact although allantoic membrane is the most favorable. Cellophane in very thin sheets forms an inner gliding surface which is smooth and the tendon heals well, but the outer surface of the tendon sheath is adherent to the surrounding structures.

Adhesions are the most frequent sequel to operations in the abdominal cavity, where the shiny peritoneal surface is denuded. The following experiments were carried out in ten dogs to determine the adhesion prevention properties of these membranes in the abdomen.

The abdomen was opened in the midline and various surfaces of the stomach, jejunum, and ileum, as well as anterior parietal peritoneum, were scarified until they presented a dull lusterless appearance and an oozing bloody surface. The surface of the ileum was then covered with one type of membrane and fixed with four interrupted sutures to a similarly denuded surface of the stomach, jejunum, ileum, or anterior parietal peritoneum. Allantoic, beef amniotic, beef cecum, and sheet catgut were used in five dogs and allowed to remain in situ from 14 to 49 days. The loops of bowel which were opposed were found adherent and could not be disengaged by gentle traction. There was no apparent difference in the results with any of the four membranes. Microscopically, there was invasion of the membrane by small round cells, and the surfaces of the bowel walls were intimately attached. Since this procedure of tacking the opposing denuded surfaces together after interposing the animal membrane did not allow for free motion of the bowel, the results were open to question. It was suggested, Dr. H. L. Johnson, of Boston, that the denuded surfaces would be prevented from disengaging themselves by the fixation sutures when the inflammatory reaction had subsided and the process of repair had begun. This would allow for an ingrowth of scar tissue from the subserosa and thus intimately and permanently fix the opposing surfaces.

Therefore, in another series of experiments the parietal peritoneum of the anterior abdominal wall was scarified and the right half was covered with human amniotic membrane while the left half was left

unprotected. Ten inches of bowel were similarly scarified and two inches of the bowel surface were covered with the same type of membrane. The dogs were sacrificed at the end of 49 and 70 days. It was found that no adhesions formed between the scarified bowel and other viscera, or to either half of the anterior abdominal wall. In another experiment the same procedure was employed except that the thick mucous covering of the amniotic membrane was removed and discarded, leaving a thin transparent glistening membrane which was placed over the right half of the parietal peritoneum. At the end of 70 days, it was found that the omentum and bowel were adherent to the denuded left half of the anterior abdominal wall and to the patch of mucus-free amniotic membrane.

The same type of experiment was carried out on two dogs with the use of allantoic membrane which was allowed to remain in situ for 65 days. The bowel and omentum were found to be adherent to the uncovered parietal peritoneum and the denuded bowel was adherent to other loops of bowel but the portion of bowel and parietal peritoneum covered with membrane remained free of adhesions.

Thus, it would appear that the presence of human amniotic membrane with its mucous surface intact prevents the formation of adhesions within the peritoneal cavity of dogs. Amniotic membrane devoid of a mucous surface does not prevent the formation of adhesions and allantoic membrane prevents adhesions only to the extent of the size of the patch of membrane used.

CONCLUSIONS

1. When allantoic and amniotic membranes, sheet catgut, and beef cecum are placed between muscle and fascia in dogs, they are absorbed and are found to be least irritating in the order named.

2. Allantoic membrane has the property of lessening the amount and degree of adhesions which form about injured nerves, tendons without sheaths and denuded peritoneal surfaces. Beef and human amniotic membranes are definitely inferior to allantoic membrane when used about nerves and tendons as an agent in lessening the formation of adhesions.

3. Amniotic membrane with its mucous surface intact has the property of preventing adhesions between denuded peritoneal surfaces. Allantoic membrane prevents the formation of adhesions between denuded peritoneal surfaces limited to the size of the membrane used.

4. Beef cecum and sheet catgut allow adhesions to form about injured nerves and tendons and in the peritoneal cavity approximately to the same degree. The results of this group of experiments show more adherence and the production of greater amount of scar tissue in the surrounding structures than in the control experiments.

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THE MODERN TREATMENT OF VARICOSE VEINS

With a Review of 285 Cases

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VARICOSE veins of the lower extremities were found in 285 cases among over 9,000 admissions to the Hutchinson Memorial Clinic of Tulane University from October, 1932, to May, 1936. Two hundred and forty-seven of these cases were females and 38 were males. Of the 247 females, 152 were treated in the Department of Obstetrics and varicose veins was simply an associated diagnosis, pregnancy being the main reason for their admission to the Clinic. The ratio of females to males in the entire series is 6.5 to 1, and the ratio of nonpregnant females to males is 2.5 to 1. Of the 247 females with varicosities, 62.6 per cent (152) accompanied pregnancy. That pregnancy is a very important factor in the production of varicose veins is further corroborated by consideration of age periods. The average age of the males with varicose veins was 48.1 years. The average age of the nonpregnant females with varicose veins was 47 years; whereas, that of the pregnant females with varicose veins was 30.6 years. The average age of the entire series of females was 36.9 years, and the average age in the entire group was 38.4 years. Thus, the average age of females is 11 years younger than the average age of males, and the average age in pregnant females with varicosities is 17.5 years younger than the average age of males. The average age of the nonpregnant female patients (47 years) and the male patients (48.1 years) is approximately the same.

The youngest male patient was 18 years of age and the oldest male patient was 79 years of age; whereas, the corresponding figures for females were 18 and 70 years, respectively. It is appreciated that the female and male groups are not exactly comparable, because in the majority of males in which the diagnosis of varicose veins was made the varicosities were the principle lesion; whereas, in most of the females the varicosities were of secondary importance. Heredity is an important factor in the production of varicose veins, as illustrated by our cases. The majority of patients stated that their father, their mother, or both, had varicose veins. A small group consisting of a mother, a sister, and a daughter all having marked varicose veins of the lower extremities had themselves noted a definite family predisposition to varicosities.

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Of the 285 cases with diagnosed varicosities in this series, only 100 were seen in the Department of Surgery. It is the routine in examining these patients to test the circulation in the varicosities of the lower extremities by three methods: (1) the Trendelenburg test,[1] (2) the Perthes test,[2] and (3) the combined tourniquet test, described by Mahorner and Ochsner[3] (Figs. 1, 2, and 3). Our test consists of having the patient walk with the extremities exposed so that the degree and extent of the varicose veins can be determined. At first no tourniquet is applied to the extremity, but after determining the type, degree, and location of the varicosities, a tourniquet is applied around the upper third of the thigh sufficiently tight to obstruct the return venous flow in the superficial veins, including the internal saphenous. The character of the varicosities is again determined while the patient is walking.

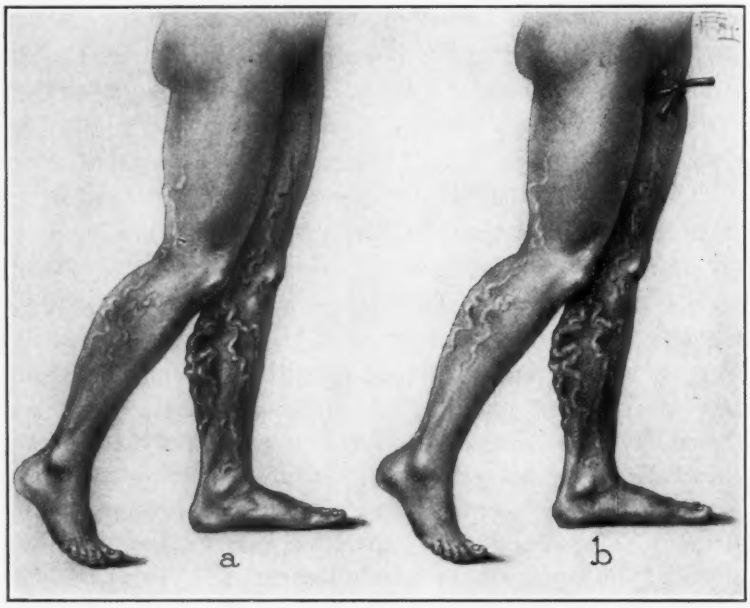

Fig. 1.—Drawings illustrating the comparative tourniquet test (Mahorner and Ochsner, Arch. Surg. 33: 479, 1936) for determining the competency of the valves of the communicating veins between the deep and the superficial systems of the thigh. The patient walks at a normal gait, and the prominence of the veins of the calf is noted (*a*). Thereafter, the tourniquet is applied to the upper third of the thigh (*b*), sufficiently tight to compress the superficial veins. Observations are made for changes in the appearance of the veins of the calf. At other times the tourniquet is similarly applied to the middle and the lower thirds of the thigh (*c* and *d*). The patient walks over the same course, and the prominence of the veins is noted. If the valves of all of the deep communicating veins of the thigh are competent and the result of the Trendelenburg test is positive, there is diminished prominence of the veins of the calf when the patient walks with the tourniquet around the upper third of the thigh. If the valves of the communicating veins are incompetent, there is little or no change because the blood spills through the incompetent communicating veins from the femoral to the long saphenous vein. When the tourniquet is below the lowest communicating vein of the thigh in which the valves are incompetent (*d*), the veins of the calf become less prominent. In this instance high ligation alone is not sufficient, and low ligation also must be performed to prevent retrograde flow through communicating veins. In *b*, slight improvement is shown over that indicated in *a*, in a case in which the blood spills through the communicating veins below the level of the tourniquet. In *c*, slight improvement (diminution in prominence) in the veins of the calf is shown, and in *d* the greatest improvement appears. If the improvement shown in *b* were as great as that illustrated in *d*, the test would indicate competency of the communicating veins of the thigh.

Similar observations are made with the tourniquet applied around the middle and lower thirds of the thigh, respectively. There will be a diminution in the degree of prominence of the varicosities when the patient walks with the tourniquet applied if the deep venous circulation is adequate. This improvement may be so marked that the varicosities disappear practically entirely, in which instance it is graded as 4. Lesser degrees of improvement are graded as 3, 2, and 1. If the improvement is as great with the tourniquet around the upper third of the thigh as it is with the tourniquet around the lower third, it is obvious that there is no leak below the main opening of the internal saphenous vein into the femoral; i.e., the valves of the communicating vein between the deep and superficial systems are competent, not permitting blood to flow from

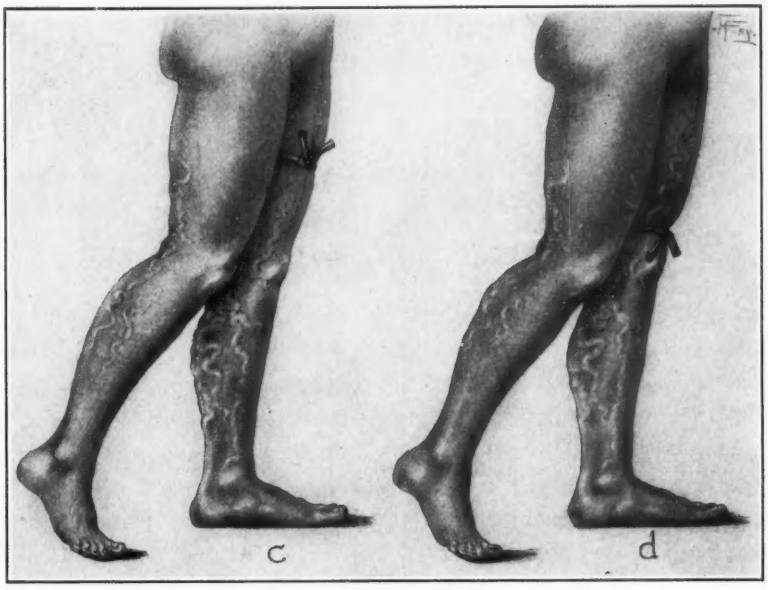

Fig. 1*c* and *d*.—See opposite page for legend.

the deep system into the superficial below the main opening of the long saphenous. In only 50 of the 100 cases herein reported was the Trendelenburg test done and so clearly recorded that there could be no mistake that the test was performed properly and interpreted properly. In few of these 50 cases the test was said to be negative, showing no incompetence of the valves of the internal saphenous vein, in spite of the fact that the patient had varicose veins of the calf of the leg. In 10 instances it was doubly positive, so that in 20 per cent of this group the valves of the communicating veins as well as those of the internal saphenous vein were found incompetent by the doubly positive Trendelenburg reaction. Our comparative tourniquet test was properly recorded for 56 extremities in 34 cases. In 19 instances the improvement with the tourniquet in the lower third of the thigh was greater than that with the tourniquet

at the middle or upper thirds, indicating that there was incompetence of the valves of the communicating veins; i.e., 33 per cent had incompetence of the communicating veins as well as incompetence of the valves of the long saphenous, in contrast to the 20 per cent obtained by the doubly positive Trendelenburg test. In 18 cases both tests were so performed that there was no question concerning the results obtained. The valves of the communicating veins were found incompetent in 4 (22.2 per cent) cases, both by the doubly positive Trendelenburg and our comparative tourniquet tests. In 2 additional cases, however, the valves were found to be incompetent by the latter test; whereas, the doubly positive reaction of the Trendelenburg test failed to demonstrate the incompetency. Thus, the comparative tourniquet test demonstrated

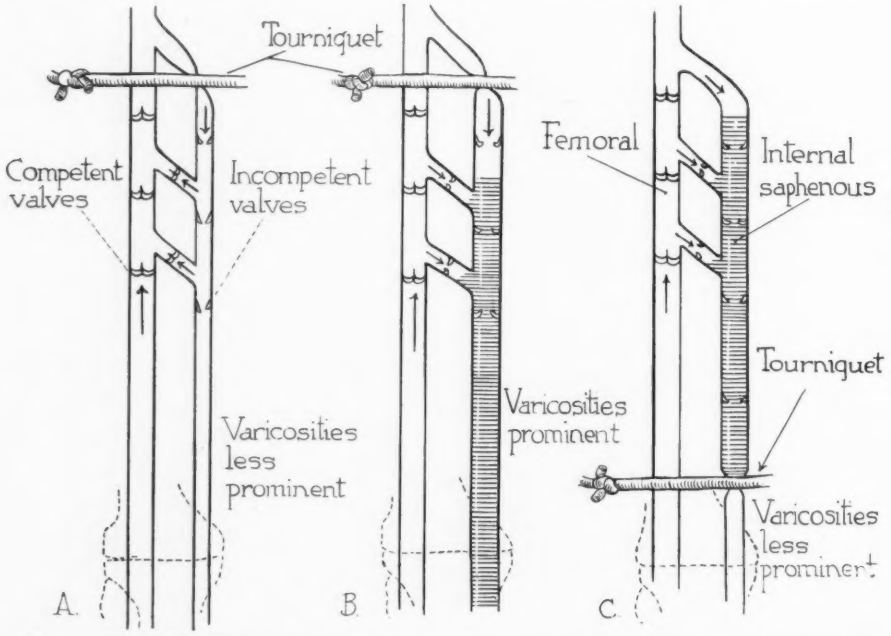

Fig. 2.—Diagrammatic illustration of the physiology involved in the comparative tourniquet test illustrated in Fig. 1. In the figure on the left (*A*), the tourniquet is applied at the upper third of the thigh sufficiently tight to constrict the internal saphenous vein and prevent the flow of blood through it at this level. The internal saphenous vein has incompetent valves, but the veins communicating between the deep and superficial systems have competent valves, and therefore when the patient walks with the tourniquet thus applied the varicosities below the level of the tourniquet become less prominent. The blood is pumped up the deep system and cannot spill back through the superficial system. In the second figure (*B*), not only are the valves of the internal saphenous system incompetent, but the valves of the veins communicating between the deep and the superficial systems are incompetent and thus the tourniquet applied in the upper third of the thigh sufficiently tight to constrict the saphenous will not stop the retrograde flow of blood through these communicating veins. Thus, the varicosities below this level when the patient walks are improved to a certain extent, but they do not entirely disappear. In the third figure (*C*), the tourniquet is applied in the lower third of the thigh below the lowest communicating veins with incompetent valves between the deep and superficial systems. In this instance, when the patient walks, there is further improvement over that of the second figure (*B*), indicating the leak through the communicating veins as well as through the main stem of the saphenous. This test shows in a statistical study that 33 per cent of the people with varicose veins have incompetent valves, not only of the internal saphenous itself, but also of the communicating veins between the deep and superficial systems. The Trendelenburg test doubly positive reaction shows incompetence of the communicating valve in only 20 per cent of a series of cases, indicating that the comparative tourniquet test is a more delicate test for estimating these additional leaks than the doubly positive Trendelenburg reaction.

incompetency of the valves of the communicating veins in 33.3 per cent; whereas, the Trendelenburg test was doubly positive in 22.2 per cent. These findings demonstrate two facts: (1) that there is a close parallelism of results obtained by the two tests, and (2) that the comparative tourniquet test is more accurate in demonstrating any leak through the communicating veins, which is of great clinical importance.

The Perthes test was recorded in 47 cases. In 46 of these, the vein below the tourniquet diminished in size when the patient walked, thus indicating indirectly that the deep veins were patent, at least at the level of the tourniquet. In 1 instance there was questionable patency of the deep veins; in other words, with the tourniquet on and the patient walking, there was no improvement and apparently a little more distention or engorgement of the superficial varicosities. From the practical standpoint with the use of the comparative tourniquet test, it is not absolutely necessary to do the Trendelenburg or the Perthes test. The comparative tourniquet test demonstrates by comparing the patient walking without the tourniquet and with the tourniquet applied at various levels whether the valves of the internal saphenous and communicating veins are incompetent, and also whether the deep veins are patent.

TREATMENT

When the Hutchinson Memorial Clinic of Tulane University first opened (1932), varicose veins of the lower extremities were treated entirely by the injection method. This was disappointing in some of the severe cases, because the sclerosis of the vein was not complete before other areas that had been injected were apparently recurring, and secondly, because even less severe cases in which the thrombosis of the varicosities was secured by treatment after a period of time had recurrences. We experimented with various procedures before arriving at a method which apparently gives the best results. Some of the procedures tried were simple ligation of the long saphenous vein with subsequent injection of sclerosing solutions, and ligations at various places in the thigh, low only, high only, and high and low, ligation of superficial veins, and ligations combined with the injection of sclerosing solutions at the same time the vein was sectioned and tied. Therefore, in the report of these results it is evident that there is no uniformity of procedure in many of the cases and that it is difficult to compare the results in the different groups. Not only have our clinical impressions been responsible for these changes during the development of our present therapy, but also comparative follow-up records show that in the cases treated by the presently used method the results are better and more permanent than in the other groups. Of the 100 cases, 8 deserted before any treatment of the varicose veins had been given, and 11 were transferred before any treatment was given, leaving 81 cases in which therapy was used. Seven of these 81 deserted during treatment before

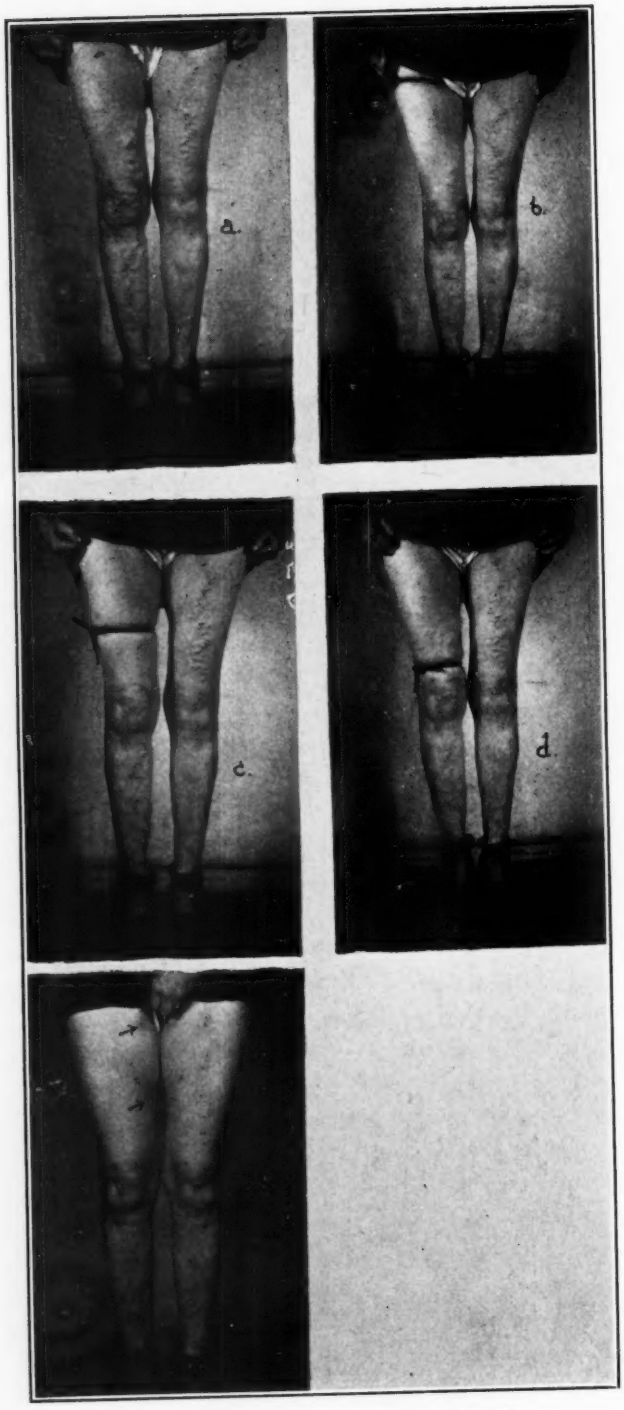

Fig. 3.—See opposite page for legend.

their course of treatment was completed. Therefore, there is a total of 72 cases in which we can report results for comparison of the various methods. In 38 of these 72 cases, ligation and section of the vein was combined with the injected treatment. In the remaining 34, the injection treatment alone was used. In employing the injection treatment, we used sodium morrhuate 5 per cent, because it had been shown previously by us[4] to be more efficient as a thrombus producing agent than other substances generally used and that it is comparatively not toxic. Recently in a few instances we have used sodium gynocardate 5 per cent and sodium soap of chaulmoogra oil, and it is our general impression that this produces a better sclerosis and more extensive thrombosis than sodium morrhuate. We have in progress some experimental work determining the value of this solution as compared to other sclerosing solutions.

In applying the injection method, we usually inject from one to three different varicosities of the patient at one time. The patient is allowed to stand and the vein is obstructed above the point of the needle just before the solution is injected. This allows the vein to collapse and the finger pressed above the point of the injecting needle permits little flow of fluid in the vein and keeps the injective solution in close contact with the intima of the vein as long as the finger compresses the vein. The compression is maintained for a minute or two, following which a compression bandage of small squares of piano felt is applied tightly over the injected area, which is removed after twenty-four hours. As a general rule, only 2 c.c. of the solution is injected in any one place, which results in thrombosis in an area varying from 2 cm. to 6 or 8 cm. From one to three varicosities are injected at one time. The patient returns at intervals of from five to seven days for repeated injections. The immediate results from this treatment have been very good, for in few instances were we unsuccessful in obtaining thrombosis completely throughout the extent of the varicosed peripheral veins.

Fig. 3.—Clinical application of the test shown in Fig. 1. The photographs were taken instantly after the patient had been walking and then stopped. In *a*, walking without a tourniquet; *b*, with tourniquet around upper third of thigh; *c*, with tourniquet around middle third of the thigh; and *d*, with tourniquet around lower third of the thigh, in each instance sufficiently tight to compress the superficial venous system. It will be noted that when the patient walks without the tourniquet the varicose veins remain prominent. When she walks with the tourniquet in the upper third of the thigh, the varicose veins though improved are not entirely gone, but when she walks with the tourniquet around the middle third of the thigh, the veins entirely disappear. Likewise, with the tourniquet around the lower third of the thigh the varicose veins are not prominent below the level of the tourniquet. The test indicates in this instance that not only is there a leak through the main opening of the saphenous, which leak is stopped by applying the tourniquet in the upper third of the thigh, but there is also a leak below this, between the upper third and the middle third, since greater improvement occurs with the tourniquet in the middle third of the thigh. Therefore, high ligation alone in this case would not cure the patient because she would have additional leaks below the main opening of the saphenous. She had high ligation and section of the vein at the upper end of the internal saphenous with injection of the distal stem, and two weeks later a ligation and section of the saphenous in the middle third of the thigh with injection of the distal stem. The lower photograph shows the patient six months after the procedure with no evidence of recurrence. The arrows indicate the scars of the operation. Ligation of the saphenous of the left leg has also been done with injection of the distal stem, but in this extremity it was necessary to ligate at only one level, as in the case of the man shown in the adjacent photograph.

LIGATION

In 38 of the patients, section and ligation of one or more veins were performed. A total of 55 ligations were done. In 24 patients high ligations were done; i.e., at the fossa ovalis and at the upper end of the internal saphenous vein above any of its tributaries. Twenty-six extremities were operated upon in these 24 patients. In 17 of the cases only high ligation of the internal saphenous on one side was performed; whereas, in the remaining 7 cases other ligations were done in addition

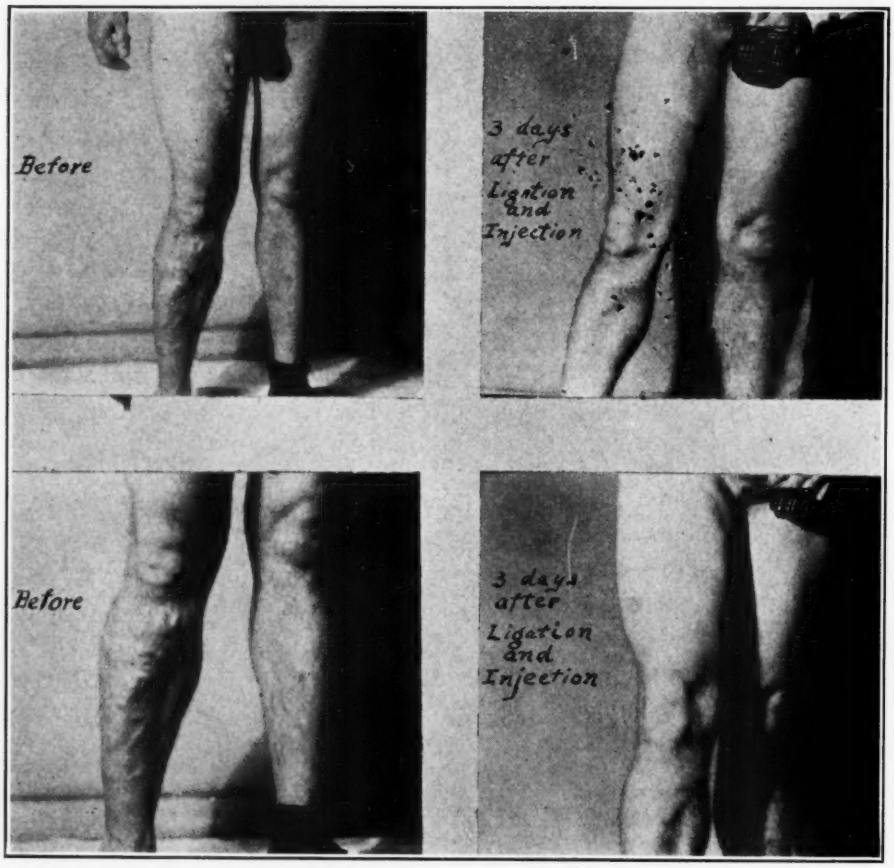

Fig. 4.—The comparative tourniquet test shown in Fig. 1 demonstrated in this patient a leak through the main stem of the saphenous with no incompetence of the valves of the veins communicating between the deep and superficial systems below the opening of the saphenous at the fossa ovalis. A single ligation and section of the upper end of the saphenous with injection of the distal stem resulted in cure. The two photographs on the left show the patient before operation and the two on the right indicate absence of varicose veins three days after the transection and ligation with injection of the distal stem. Compare with woman shown in Fig. 3, page 894.

to high ligation on one side. In 3 cases, in addition to the high ligation, the internal saphenous was also ligated in the lower third of the thigh. In 14 patients the only ligations did not include high ligation of the internal saphenous, but other operations to section and ligate veins (the internal saphenous in the middle or lower third of the thigh, or

superficial tributary veins of the internal saphenous system) were done. This group of 14 patients in whom ligations were done below the upper end of the internal saphenous channel not combined with ligation of the internal saphenous at the fossa ovalis form an excellent group for comparison of results. We have had the same experience as Faxon,[5] who emphasizes so strongly that to prevent recurrence ligation must be done high on the internal saphenous. In one case the external saphenous system was ligated below the knee, and in this instance a retrograde flow through the main opening of the external saphenous vein into the popliteal vein was demonstrated by the comparative tourniquet test. This incompetence was detected by the fact that the vein did not improve when the patient walked with the tourniquet on the thigh at any of the usual three levels, but did improve when the tourniquet was placed below the knee and constricted the main stem of the external saphenous vein.

METHODS OF MANAGEMENT

On examination of the patient with varicose veins, if the valves of the internal saphenous system are found to be incompetent, the Trendelenburg test is positive and the comparative tourniquet test shows improvement when the patient walks with the tourniquet at the upper, middle, or lower third of the thigh, then we recommend ligation. We do not perform ligation in mild cases of varicosities or those cases in which Trendelenburg test is not positive or in which it is questionable. In mild cases of varicosities we still adhere to the injection treatment alone, and feel that if there is a great chance of recurrence the patient is at least temporarily relieved and that, if necessary, a more radical procedure in attacking the varicose veins can be performed. At the present time we feel that ligation and section of the internal saphenous system should be done high, above all of the tributaries of the internal saphenous system, because we have observed recurrences when ligation of the saphenous was performed in the middle and lower thirds of the thigh, and we believe that there is less chance for recurrence through collateral veins if the ligation is done above all collateral veins. In addition to high ligation of the internal saphenous on every extremity with moderate or severe varicosities, additional ligation and section is done below the level of the lowest communicating vein in the thigh with incompetent valves in those cases in which the comparative tourniquet test shows incompetence of the valves of the veins communicating between the deep and superficial system (Figs. 1, 2, and 3).

OPERATION

No special preoperative preparation of the patient is necessary. The field of the operation is prepared by shaving and by the usual skin preparation. The femoral artery is palpated at Poupart's ligament and the

line of the artery may be marked with gentian violet. A longitudinal incision is made beginning 2 cm. medial to the line of the femoral artery and from 2 cm. below Poupart's ligament downward for a distance of 4 cm. After a little experience it is possible to come directly on to the internal saphenous vein near where it opens into the femoral vein at the fossa ovalis. Not infrequently the external pudendal vein will be recognized and it is necessary, as a general rule, to ligate this separately or to ligate the long saphenous above this tributary. The long saphenous is isolated and clamped with a curved hemostat and 2 c.c. of 5 per cent sodium morrhuate solution is injected into the distal segment. Another hemostat is applied just distal to the point of injection and the vein is severed between these two hemostats. The proximal stump is then transfixed and ligated, and another ligature without transfixion is put on the stump above the area of transfixion. This precautionary measure is done theoretically to prevent the propagation of a thrombus which may have originated at the catgut transfixing the vein. Because we have had one ligature slip before the present routine of transfixing the vein, we now transfix the vein and since doing this have had no difficulty whatsoever. The inferior stump is then transfixed and ligated, but it is not necessary here to apply an additional ligature around the vein distal to the transfixion because a thrombus in the lumen of this vein is desirable. We use, as a general rule, chromic No. 1 or No. 0 catgut for this ligation. The subcutaneous tissues and skin are closed with interrupted sutures. After applying a firm dressing the patient is allowed to remain on the table for one hour. They are instructed then to go home, and to rest, either in bed or in a chair, but not to remain entirely in bed for more than a few hours at a time. Further, they are instructed to get up and to move around the room with the belief that this encourages the circulation in the deep veins of the lower extremities and helps to prevent propagation of a thrombus through the collateral veins into the deep system. We find that the majority of the superficial veins after this ligation become thrombosed soon after the operation and that the thrombus progresses downward gradually within the next few days, extending anywhere from the middle third of the thigh to the lower part of the middle of the calf of the leg. The wound is dressed on the third or the fourth day and the sutures are removed after the eighth to the tenth day. We have frequently found some induration in and around the wound in the inguinal region and have attributed this to leakage of the sodium morrhuate into the wound while injecting. In those cases where our comparative tourniquet test shows that the patient has a leak not only through the main opening of the internal saphenous vein, but also through the communicating veins from the deep to the superficial system, we ligate the internal saphenous above all its tributaries and also in the middle or lower third of the thigh, the site depending upon the results of the comparative tourniquet test. This

second ligation is done notwithstanding the fact that a thrombus may have started in the distal stump and propagated beyond this level. These thrombi disappear or wash out after a period of time if the pressure in the venous system is high. They obstruct the lumen of the vein temporarily, but the thrombus may later disappear, possibly because of abnormally high pressure arising because of incompetence of the valves of the veins communicating between the deep and superficial systems or through processes of repair. To prevent this regeneration, secondary ligation of the stem of the internal saphenous is done in the middle or lower third of the high below the lowest communicating vein between the deep and superficial systems of the thigh with incompetent valves, as demonstrated by our test, from ten days to three weeks after the first ligation. Other secondary ligations may be done for marked varicosities in collateral channels and these are frequently found on the anterior surface of the thigh subcutaneously, a vein tributary to the external circumflex iliac or internal saphenous, i.e., the external superficial femoral. Additional ligations may be made posteriorly on the thigh, channels running from the gluteal veins or veins in the gluteal region. Independent ligation of these trunks is frequently necessary and at the time of ligation injection of sodium morrhuate is made into the distal stump. Varicose veins in these regions have a peripheral retrograde flow and the ligation should be made at the highest level to produce obliteration of the entire vein. Occasionally posteriorly on the lower third of the thigh varicose channels are found in which the flow is toward the external saphenous system. If ligation is indicated here, on theoretical grounds it should be done low on the varicosity and the vein injected above the point of section to prevent a thrombus from entering the popliteal vein. In rare instances we have found that the external saphenous system itself was varicosed, not by virtue of the connections with the internal saphenous system, but because of incompetent valves in the main stem of the former, which opens into the popliteal. This is diagnosed by the comparative tourniquet test. When the veins of the calf which comprise the external saphenous system do not collapse when the tourniquet is placed even at the lower third of the thigh and the patient walks, but do disappear when the tourniquet is placed immediately below the knee, it is evident that the varicosities of the calf neither are due to a direct leak through the communicating veins from the deep to the superficial system in the calf nor are from a retrograde flow from the internal saphenous system in the thigh, but are the result of incompetence of valves of the external saphenous system. If the varicosities were due to leaks through the communicating veins between the deep and superficial systems in the calf, there would be no improvement even when the tourniquet is placed below the knee. Ligation and section and simultaneous injection of sclerosing solution do not take care of all of the varicose veins in every instance. Occa-

sionally, a few varicosities remain on the calf or the thigh which should be injected with sclerosing solutions. We find that after ligation the tension or pressure in the vein itself is greatly reduced and that it is easier to produce thrombosis and the incidence of recurrence is diminished.

ULCERS

The presence of varicose ulcers changes the treatment routine for varicose veins. Of the cases in our series, 29 had varicose ulcers. Twenty-one of these were females and 8 were males. This incidence of 29 per cent ulcers is a very good indication of the severity of the cases. Many of these varicose ulcers were long standing and large. Some had been present for as long as twenty years. The youngest male with a varicose ulcer was 33 years of age and the oldest male was 72. The average age of the males with varicose ulcer was 50.4 years. The youngest female was 32 years of age and the oldest female was aged 70 years. The average age of the females with varicose ulcers was 49⅓ years. Veins are not ligated in the presence of an infected ulcer. The ulcer must be treated conservatively and must be entirely healed and free of infection before ligation is attempted. Though we have ligated a few veins in the presence of ulcers which were almost healed, we believe that ligation in the presence of infection below is not without danger because of the possibility of propagation of the infection. In our series, we have had no operative fatalities and no emboli, either fatal or merely small infarctions of the lungs. The treatment of the ulcer depended upon its type and appearance when the patient was first seen. If it was acutely inflamed or badly infected, the patient was treated with bed rest, elevation of the involved extremity, and application of "hypertonic" saline dressings. When the acute infection subsided and healthy granulation tissue began to appear, the ulcer was treated by compression dressings or an Unna's paste boot. In cases in which compression was used, sterile xeroform gauze (5 per cent xeroform in white vaseline) was applied directly to the ulcer, and this was covered with sterile plain gauze and fixed in position with adhesive plaster. A moist marine sponge (sterilized by immersing for one week in $\frac{1}{1000}$ bichloride solution and washed thoroughly with sterile water) is then applied to the covered ulcer and firmly compressed against it by applying a snug circular bandage. Occasionally a rubber bath sponge was used instead of a marine sponge, but we found that the marine sponge gave a more even compression. The patient returned every three to five days for renewal of the dressing. Unna's paste boot was found even more effective in some cases. Care should be taken to see that it is properly applied, and it can remain on for from ten days to three weeks. It was not always possible to promote healing by these conservative methods. Five patients were referred to Neal Owens, in charge of the Plastic Surgery Department. All of these were hospitalized in

order to excise badly scarred ulcer base and to perform skin grafts. In 3 instances Davis grafts alone were done; in 1 of these it was repeated. In 2 instances Wolfe-Kraus as well as Davis grafts were used. In spite of the fact that in every instance the ulcer healed, they did not all remain healed. In 3 cases ulcers broke down one or more times. It has been our experience that ulcers of the leg due to varicose veins, as a general rule, occur in dirty, indolent, indifferent, careless patients. These characteristics predispose to a recurrence and we have begun to regard the bad leg ulcer as almost hopeless for a permanent cure, and a lifelong charge on the institution. It is not so much the pathology as the indolence of the patients who repeatedly provide an activating cause which prompts the expression of this note of discouragement in these cases.

RESULTS

It was possible to reexamine 25 patients who had finished the injection treatment with no operative procedure from six months to three and one-half years previously. Twenty patients who had been treated by ligation and injection at one or more levels from six months to eighteen months previously were reexamined. Of those treated by injection method alone, there were 40 extremities in the 25 patients, 15 patients receiving treatment in both lower extremities. There was recurrence of varicosities in 23 (57.5 per cent), no recurrence in 17 (42.5 per cent). In fairness to the method, it was noted by us as by others that the subjective results were better than the objective; i.e., a patient may feel that the varicose veins have remained well, although there are definite varicosities present. In general, the patients feel more comfortable and do not feel fatigue in the extremities as quickly, and other symptoms have not returned with the same intensity as when originally present. It may be that the pressure in these varicosities is less and that partial thrombosis above these areas has diminished to some extent the volume and rapidity of back flow in the internal saphenous system. Five of the 25 patients who had ligations at one or more levels of the internal saphenous system had ligations in both lower extremities. There were 25 extremities in which reexamination was possible from six months to two and one-half years after the procedure. Eleven of the extremities had ligations only at the upper end of the internal saphenous vein with simultaneous injection of a sclerosing solution. Two of these recurred (18 per cent), 9 (82 per cent) remained well (in contrast to 42.5 per cent with the injection treatment). Of 12 ligations of the internal saphenous in the middle or lower third of the thigh without ligation of the same saphenous at the upper end, 6 had recurrence of varicosities (50 per cent), and 6 (50 per cent) remained well. Only 2 cases were reexamined where high and low ligations were performed on the same saphenous vein. One of these had recurrence, but 2 is too few for comparison.

SUMMARY

Of 247 cases of varicose veins, approximately 60 per cent were in pregnant females.

Pregnancy and heredity are both important etiologic factors in the development of varicosities.

An accurate knowledge of competency of the valves in the superficial and communicating veins is essential to determine the type of therapy to be used in varicosities, and also for prognostication.

The comparative tourniquet test previously described by us is the most sensitive and most accurate test for determining incompetency of the valves of the veins communicating between the deep and superficial veins, and therefore the most important for determining the type of therapy.

Injection of sclerosing solution alone in the treatment of varicose veins resulted in recurrence in 57.5 per cent of instances; whereas, high ligation and section of the saphenous with simultaneous injection of sclerosing solution resulted in recurrence in only 18 per cent of instances.

The physiologic reasons are stated why it is essential to ligate low as well as high in the cases where the comparative tourniquet test demonstrates incompetence of the valves of the veins communicating between the main deep veins and the superficial systems.

Varicose ulcers are treated conservatively before radical measures to cure the varicosities are instituted.

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VARICOSE VEINS TREATED BY COMBINED LIGATION AND INJECTION

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THE present popular method of treating varicose veins by the injection of sclerosing solutions appeals to both the physician and the patient in that it is simple and safe, inexpensive, and ambulatory. Experience has taught, however, that when employed alone the method is followed by frequent recurrences, even greater than those following the older operations of ligation, stripping, and excision. To avoid these recurrences and to facilitate primary obliteration by injections, several ligation procedures are now employed as adjuncts to the injection method.

When one recalls that varicose veins were ligated as early as the seventh century, that an injection method was used as early as 1853, and that even ligations and injections were combined in 1904, the progress in the therapy of varicose veins seems hardly remarkable. Nevertheless, improvement in the solutions used for injections and a better understanding of the principles of ligation have made modern treatment highly satisfactory. Thus, the older toxic coagulants used for injections have been replaced by nontoxic sclerosing solutions and the point of saphenous ligation has been progressively advanced upward to reach finally the femoral junction itself, above all the saphenous tributaries.

No attempt will be made to review the vast amount of literature, that has accumulated on the treatment of varicose veins. An exhaustive historical review may be found in E. Edwards'[1] article in *Surgery, Gynecology and Obstetrics* for December, 1934. Articles by De Takats,[2] Homans,[3] Faxon,[4] Ferguson,[5] and the paper of E. Edwards referred to above are pertinent to the thesis of this paper, "The Treatment of Varicose Veins." Early in the return to popularity of the injection method, De Takats advocated ligation of the saphenous high in the upper thigh preliminary to injections, whenever the saphenous was very dilated. He claimed by this procedure to prevent embolism and to increase the ease of subsequent sclerosis by injection due to a reduction in downward venous pressure. A third and by far more important indication for some ligation procedure, accessory to the injection method, soon became apparent as the percentage of recurrence following the injection method alone was found to be much greater than originally reported. These recur-

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rences were attributed chiefly to recanalization of the veins from excessive downward venous pressure and an associated ligation procedure could be expected to correct the trouble. When, however, the ligation was performed high in the thigh after the manner of De Takats, recurrences were still frequent, due to elongation of the saphenous stump left by the operation, or to dilatations of the tributary veins above the ligation site. Faxon, Edwards, the writer, and others following Homan's suggestion raised the ligation site to the very femorosaphenous junction, ligating the vein flush with the femoral and dividing the branches encountered at this point. With the recanalizing influence of the downward venous pressure eliminated, with tributaries which might

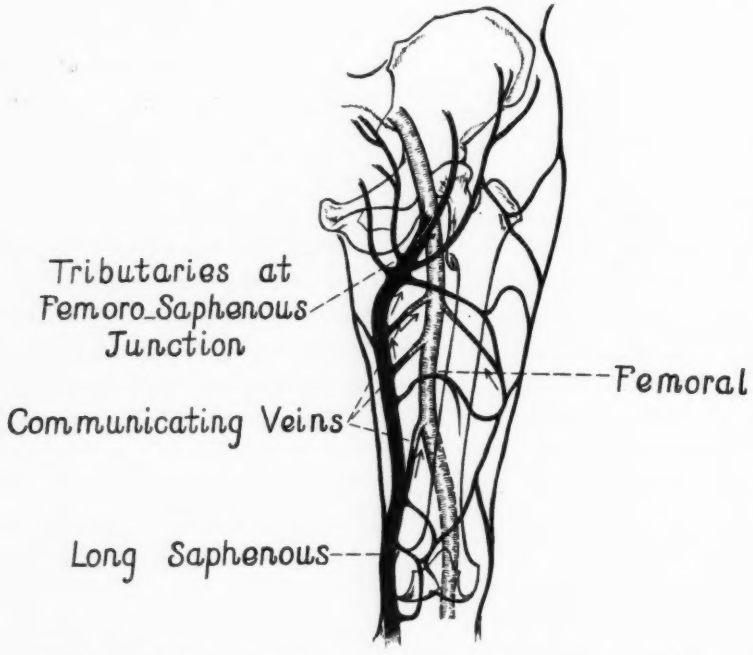

Fig. 1.—Relationship of long saphenous to deep venous system.

form collateral circulation interrupted, and without a stump which might elongate, the results of the injection method could be expected to be much better. Statistics are rapidly accumulating to indicate this to be the case. However, high ligation and injection, alone, often do not prove adequate for "blow-out" of the long saphenous in the mid or lower thigh. The explanation for this is given later, but suffice it to say now that such cases require multiple ligation procedures as described by Ferguson,[5] each case being individualized and ligation procedures performed at the femorosaphenous junction or at any other indicated "blow-out" sites.

In the combined method of treating varicose veins, injections are made through the opened vein at the time of the ligation or subse-

quently by the usual technique, according to individual preference. The stages in the change of modern conception of the treatment may be summarized as follows:

1. Revival of the old injection method employing nontoxic sclerosing solutions instead of toxic coagulants.

2. Revival of ligation preliminary to injection.

3. Advance upward of the ligation site to the femorosaphenous junction with division of all tributaries at this point. Simultaneous or subsequent injection of the distal segment.

4. Ligation of all "blow-out" points with simultaneous or subsequent injections.

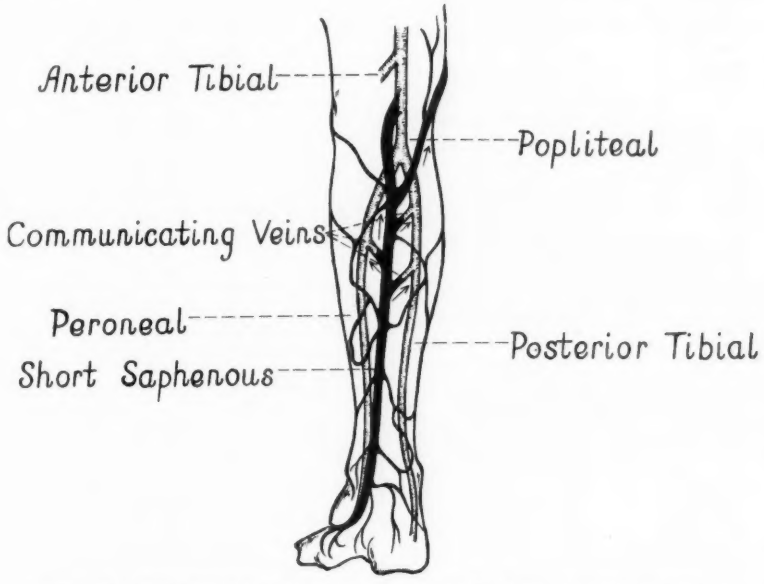

Fig. 2.—Relationship of short saphenous to deep venous system.

5. Controlled injections, the extent of the inflammation being limited by a secondary lower vein ligation.

Experience rapidly taught me, as it did others, that no two cases are alike, and that a procedure indicated for one would be of no value or would even be contraindicated for another. In general, I employ the injection method alone for simple cases with a negative Trendelenburg test, and radically high ligation and simultaneous injection, whenever the saphenous is dilated high in the thigh and whenever Trendelenburg's test is positive. Separate ligations with or without simultaneous injections are also made at all "blow-out" points lower in the leg. In addition, whenever indicated, I have limited the segment of vein affected by the sclerosing solution by injecting between two ligated points. I will try as briefly as possible to describe the principles and technique employed.

Although throughout this paper emphasis is placed on the combination of ligation and injection, it is not to be inferred that ligation is an invariable adjunct to the injection method. A large percentage of cases is still handled by injections alone. Even some large varicosities respond to the simple injection as long as they are not in intimate connection with an incompetent saphenous trunk or an incompetent communicating vein.

NORMAL ANATOMY

Large arteries carry a more or less constant supply of blood to the lower extremity to be returned from the leg by two systems of veins, a deep system accompanying the principal arteries, and a superficial system having no arterial analogue. This superficial venous system is

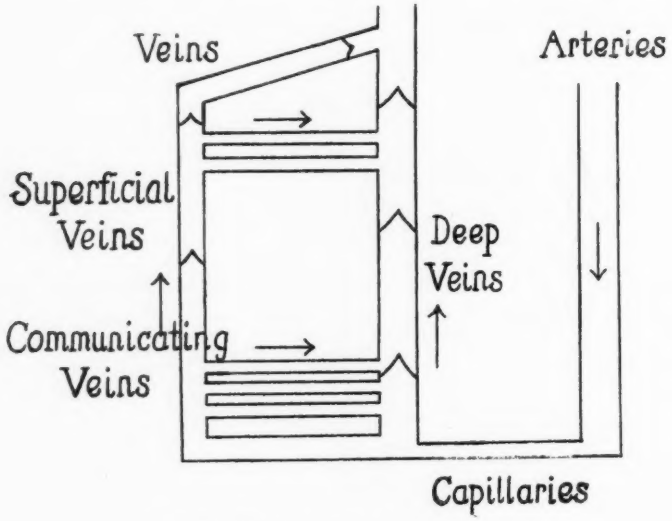

Fig. 3.—Normal venous circulation.

apparently not indispensable, as suggested by the lack of dire consequences when a considerable portion of it is destroyed in the treatment of its chief disease, varicosity. The following characteristics of the venous systems of the lower extremity are important in our discussion.

1. The veins of the superficial system are connected by important communicating veins with the deep system. The communications are frequent in the lower leg, especially about the ankle, calf, and knee. In the thigh there are at least two important communications in addition to the femorosaphenous junction, one about one-third way up and the other about one-half way up the thigh.

2. The veins of both systems are supplied with valves with cusps arranged to maintain the upward position reached by the column of blood. The valves of the communicating veins normally allow the blood to flow from the superficial to the deep veins.

3. The veins of the superficial system, all branches of the long and short saphenous, are relatively poorly supported by soft parts and readily dilate to become varicose. The short saphenous passes under

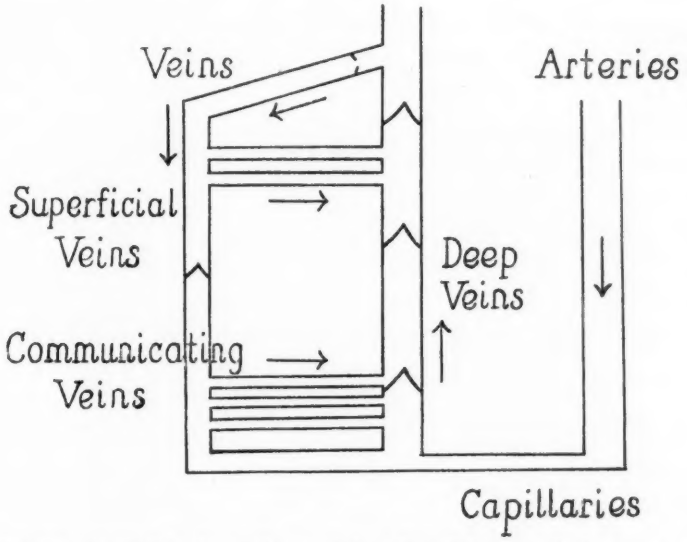

Fig. 4.—Venous circulation in varicose state. Trendelenburg test may or may not be positive.

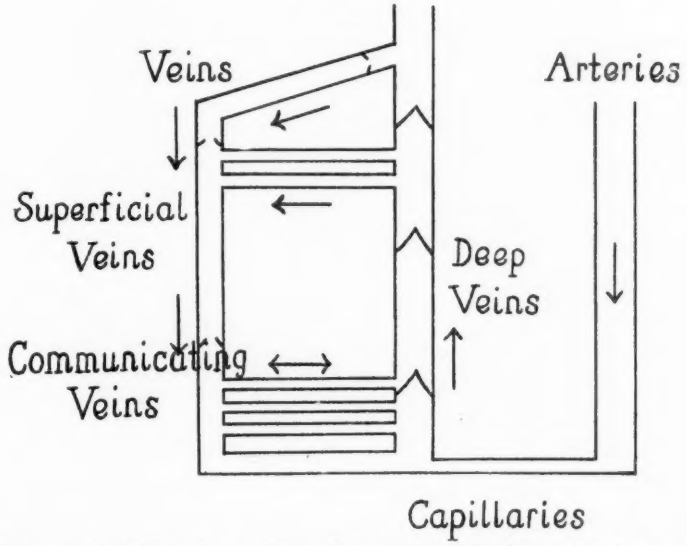

Fig. 5.—Venous circulation in varicose state, communicating veins incompetent and Trendelenburg test often doubly positive.

the deep fascia for several inches before entering the popliteal, and this fascial support makes it somewhat less prone to give away than the long saphenous.

4. The short and long saphenous are connected by a branch on the inner side of the leg above the knee. Thus a "give-away" of the long saphenous may lead to varicose formation in the short saphenous.

THE VARICOSE STATE

The veins of the superficial system, possibly hereditarily predisposed, readily enlarge when subjected to factors of pressure, obstruction, injury, hormonal influence, or infection. Associated with the dilatation is an incompetent condition of the vein valves so that the flow of blood is retrograde in the upright position. Blood passes down instead of up the short or long saphenous, then through the communicating veins of the lower leg into the deep system and up through the popliteal and femoral to the thigh. There is then usually a spill over into the long saphenous again, due to the incompetency of the valve at the femorosaphenous junction. Further, this varicose state may involve the communicating veins, so that the blood may pass abnormally from the deep to the superficial veins at one or more points in the leg in addition to the spill over at the femorosaphenous junction.

EXAMINATION OF THE PATIENT AND PLAN OF TREATMENT

After the routine physical examination has been made, the patient is asked to stand up before a good light for accurate study of his varicose condition. Often a plain or infra-red photograph is taken for "before and after records." Inspection and palpation are then employed to locate the main varicose trunks, and the uppermost point of enlargement of these trunks is recorded as the "give-away" or "blow-out" site. If the varicosities are small, no special tests are performed. Such cases require simple injections only. Some of my experiences with the injection method alone are described in previous articles.[6, 7] It is well to remember that even these small veins have a tendency to recur and the patient should return at stated intervals for a considerable time for observation.

We are concerned in this paper principally with large varicose veins or cases of recurrence following previous injection or operative attempts at cure. The long saphenous may or may not be visibly dilated in the thigh, but the intelligent use of the tourniquet, bearing in mind Trendelenburg's principles, will always demonstrate the nature of the pathology present.

A positive Trendelenburg test with the tourniquet applied high in the groin demonstrates a "spill-over" from the deep to the superficial system at the femorosaphenous point and at once indicates that the case should be ligated, as well as injected, at this site, if rapid recanalization and recurrence are to be avoided. Often the test will be positive with respect to varicosities in the lower leg even though the long saphenous does not seem to be dilated on inspection or palpation. It is important to bear this in mind in the obese, in whom an unsuspected incompetent long saphenous is the chief cause of therapeutic failure.

Should the case present dilatation of the long saphenous lower than the femorosaphenous junction, the suspected "blow-out" site is tested by a modification of the Trendelenburg test. The extremity is raised and the finger used to compress the uppermost part of the dilated vein. Still compressing the vein, the leg is lowered. A reflux of blood downward as the finger is removed indicates incompetency of the vein valve at this point with probable competency of the valves above. Such

Fig. 6.—Infra-red photograph of a case of varicose veins.

"blow-out" points occur often in the mid thigh, lower third of the upper leg, and at the knee level. "Blow-outs" at these points require simple ligation and division of the vein at the "blow-out" level followed by the injection of the related varicosities. The operation will be above the highest incompetent valve and below the lowest competent one. Ligation higher in the groin at the femorosaphenous junction will fail entirely here, as can be demonstrated by performing Trendelenburg's test with the tourniquet in the groin in the usual fashion.

Cases are also encountered which present a "blow-out" in the mid or lower thigh as well as some incompetency at the femorosaphenous point. Operation here requires a double procedure, high ligation and injection at the femorosaphenous junction and separate ligation of the lower "blow-out" point.

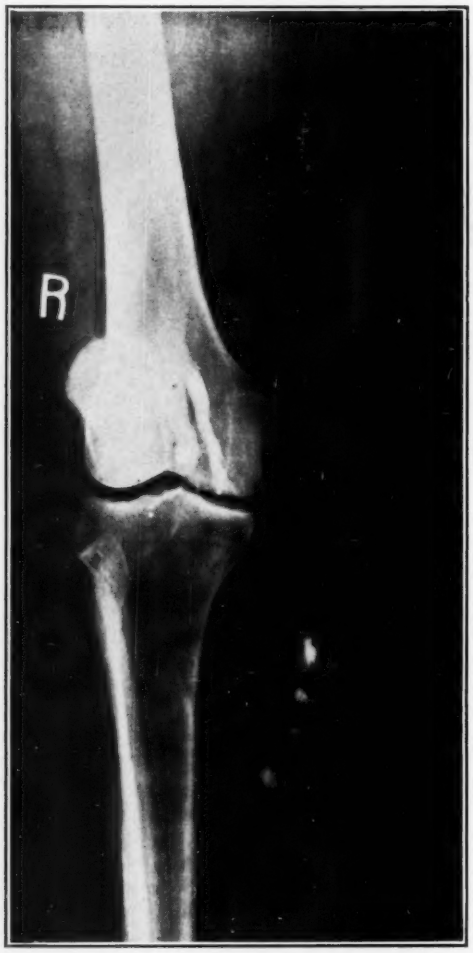

Fig. 7.—A radiograph with hippuran demonstrating an unsuspected incompetent long saphenous. Visible varices only in lower leg.

Varicosities of the calf and lateral aspect of the leg require careful inspection and palpation of the short saphenous, as a "blow-out" may exist which requires ligation and division in the popliteal space. Ligation of the long saphenous at any point in the leg cannot be expected to influence varicosities of this vein, which joins the femoral directly in the popliteal triangle.

Having decided upon the "blow-out" sites, I then usually study the venous tree below this point to decide how extensive the inflammatory

reaction is apt to be if the proximal end of the vein is injected at the time of ligation. Usually a second point for ligation is chosen to limit this reaction as discussed later.

I lay little emphasis on the test for patency of the deep veins, although this must of course be known before any method of treating varicose veins is adopted. This matter may be summarized as follows:

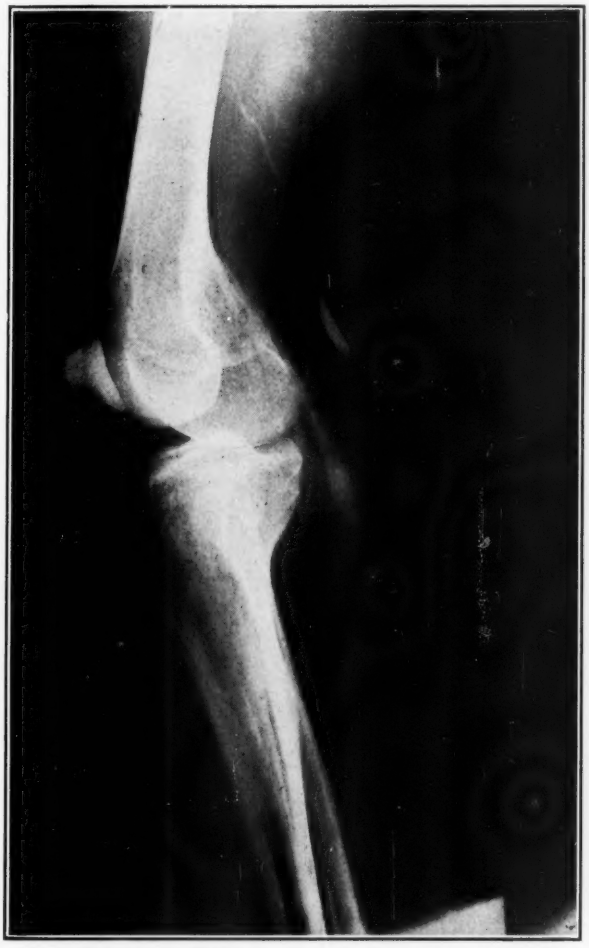

Fig. 8.—Radiograph with hippuran. The Trendelenburg test is doubly positive. A long vein communicating with the deep and superficial system is seen.

1. Deep Veins Patent.—No past history of "milk leg"; no edema of leg; tight elastic stocking worn with comfort; negative Perthes test.

2. Deep Veins Occluded.—Compensatory enlargement of veins across lower abdomen; persistent leg edema and leg fibrosis; positive Perthes test; past history of "milk leg."

The examination of the patient is not complete unless the relation of all the varicosities to the main venous trunk has been determined. Often, percussing a varix, while palpating the main trunk above it,

will demonstrate their relationship. In very puzzling cases, x-ray visualization of the varicosities, after the injection of hippuran, is of value. Undiluted hippuran when injected into a varicosity for x-ray visualization frequently thromboses the veins. This simply lessens the number of injections the patient will need subsequently, but it is well to advise the patient what it to be expected. The procedure should not be used if the deep veins are occluded.

Fig. 9.—Radiograph with hippuran. A case showing secondary "blow-out" point below knee. Accessory branches are seen to be the cause of secondary dilatation.

INDICATIONS FOR COMBINED LIGATION AND INJECTION

1. All positive Trendelenburg cases. The operation is performed at the femorosaphenous junction. Often the saphenous is also divided just above the knee to limit the inflammatory reaction from injection.

2. All cases with well-defined "blow-out" sites lower in leg. The operation is done in mid thigh, lower thigh, or wherever the "blow-out" occurs.

3. Varicosities of the short saphenous require ligation in the popliteal space.

4. Cases presenting a doubly positive Trendelenburg theoretically demand division of all communicating veins as well as femorosaphenous ligation and injection. Practically it often suffices to ligate and

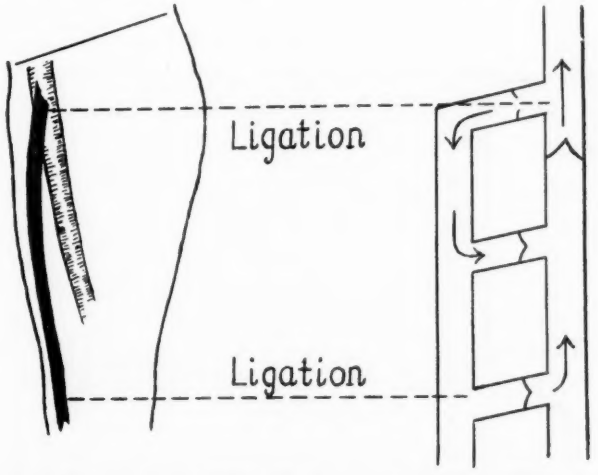

Fig. 10.—Diagrammatic illustration of cases requiring ligation. All cases presenting a positive Trendelenburg. Ligation and injection at femorosaphenous junction. Secondary ligation lower in leg to limit inflammatory reaction from injection.

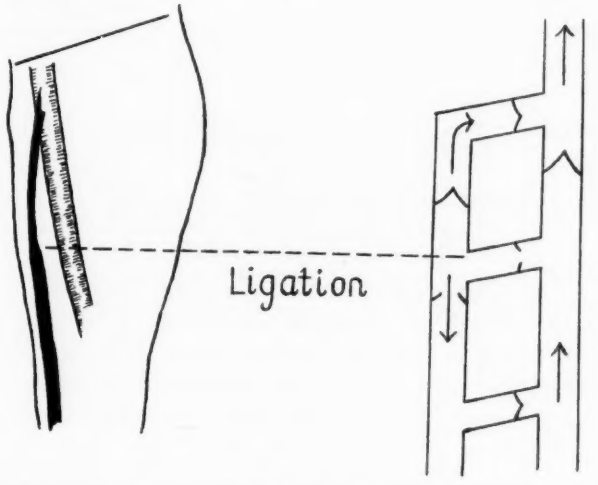

Fig. 11.—Diagrammatic illustration of cases requiring ligation. Cases presenting "blow-outs" below femorosaphenous site. Figures illustrate ligation of "blow-out" in mid thigh, above incompetent valve and below last competent valve.

inject at the femorosaphenous point and at each "blow-out" site lower in the leg, dividing communicating veins encountered in the operative areas. In persistent cases the operation recently described by Faxon[8] may be performed. The deep fascia is incised extensively and the communicating veins divided as the fascial flaps are dissected back.

5. Cases exhibiting a positive Trendelenburg and a secondary "blow-out" site lower in the leg. In addition to ligation and injection at the femorosaphenous junction, another ligation lower in the leg at the secondary "blow-out" site is required.

Contraindications to Combined Ligation and Injection.—These are relatively few. Persistent occlusion of the deep veins constitutes a definite contraindication. Nephritis, hyperthyroidism, cardiac disease may make the procedure inadvisable. Pregnancy is not a contraindication. To the contrary, high ligation influences favorably even varicosities of the vulva in pregnancy.

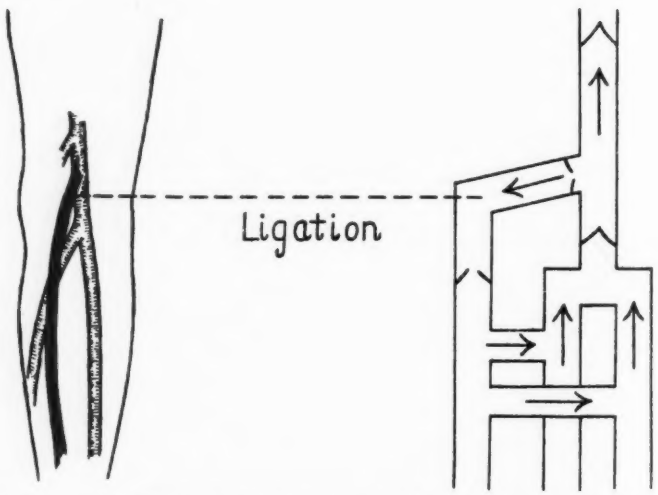

Fig. 12.—Diagrammatic illustration of cases requiring ligation. "Blow-out" of short saphenous. Ligation in popliteal space.

THE TECHNIQUE OF LIGATION OF "BLOW-OUT" BELOW THE FEMOROSAPHENOUS JUNCTION

This is a simple office procedure. The site of the proposed incision is marked with the patient standing. Acriflavine, 4 per cent, followed by tincture of iodine is used for this purpose, a black cross resulting. With the patient recumbent, the operative field is further painted with tincture of iodine, preserving the black cross of the proposed incision. The area is infiltrated with 1 per cent novocaine and the vein is cut down upon, through a transverse incision about 1½ inches wide. Even in the lower thigh and leg, the saphenous is under the superficial fascia which must be divided. It is important not to mistake a small superficial branch for the larger vein under the fascia. Several centimeters of the vein are resected, the distal end being injected with sodium morrhuate solution, if desired, after the manner described below under femorosaphenous ligation. Any collateral veins in the field of operation are divided. A careful search is made for a communicating vein to the deep system and same divided if found. Interrupted

sutures are used to obliterate the dead space and the skin closed with dermal. An Ace leg bandage is used to bind the leg and hold the dressing in place. The patient is allowed to get up at once and provided with a few codeine tablets for pain. He is encouraged to resume his daily routine. At subsequent visits, the varicosities, which have not spontaneously thrombosed following the ligation, are injected in the usual manner.

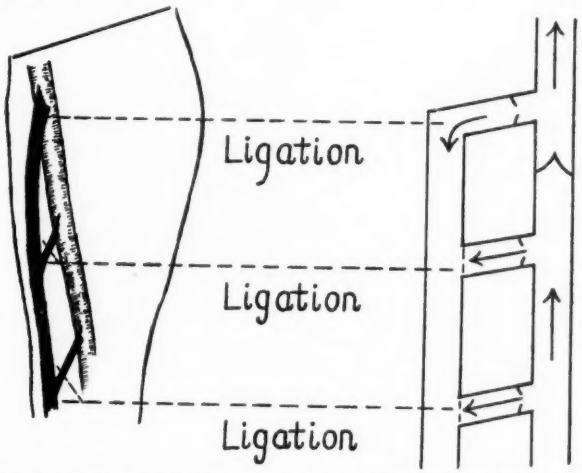

Fig. 13.—Diagrammatic illustration of cases requiring ligation. Cases exhibiting a doubly positive Trendelenburg require ligation at femorosaphenous junction and all communicating veins lower in leg.

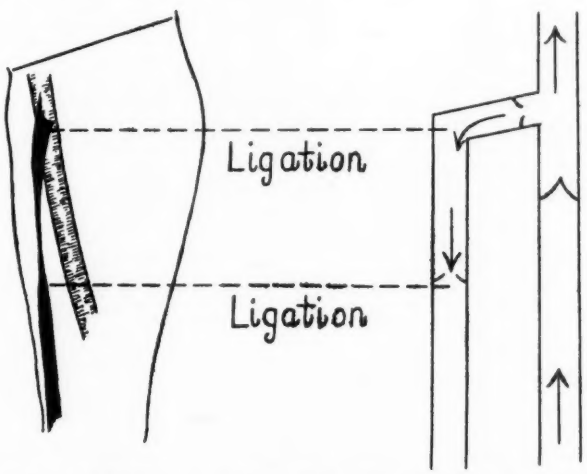

Fig. 14.—Cases exhibiting a positive Trendelenburg and a secondary "blow-out" lower in leg. In addition to ligation and injection at femorosaphenous junction another ligation lower in leg at secondary "blow-out site is required.

THE TECHNIQUE OF LIGATION AND SIMULTANEOUS INJECTION OF THE SAPHENOUS AT THE FEMOROSAPHENOUS JUNCTION

This is a somewhat more formidable procedure when properly done and is best performed in the hospital operating room. The landmarks are the spine of the symphysis and the pulsating femoral artery. The

femorosaphenous junction is about 1.7 cm. below the level of the spine of the symphysis as shown by Edwards[1] in numerous dissections. I make a transverse incision extending from the femoral pulsation mesialward, about 1 cm. below the presumed site of the femorosaphenous junction, or about 3 cm. below the level of the spine of the symphysis, thus approaching the vein a little below the femorosaphenous point. To operate directly over the junction is needlessly confusing.

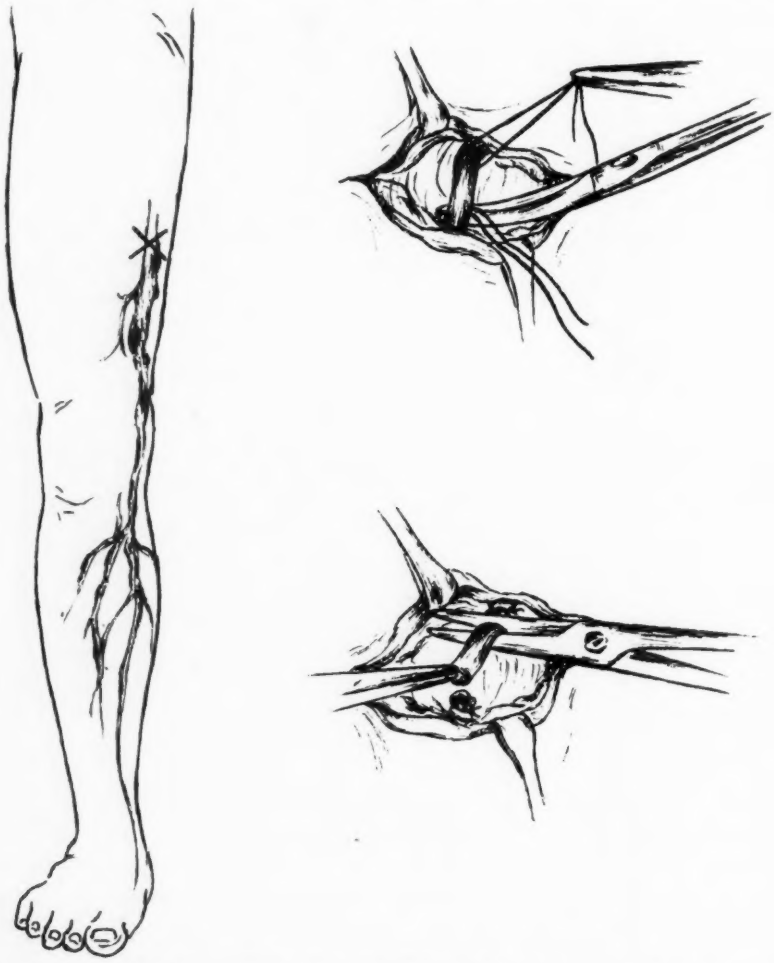

Fig. 15.—The technique of ligation of "blow-outs" below the femorosaphenous junction. With patient standing the site is marked with acriflavine (4 per cent) and iodine. Note that the vein is often under the superficial fascia.

The skin edges are retracted with Allis clamps or small blunt retractors, and the dissection deepened sharply to the superficial fascia which must always be identified and divided. A little blunt dissection usually readily exposes the long saphenous, resting in fatty tissue under the superficial fascia, and proximal to the fascia lata of the thigh. The vein is separated from its bed over several centimeters and plain cat-

gut sutures are placed but not tied around the upper and the lower ends of the freed segment. A nick is made in the lower end of the vein between two mosquito clamps, and a transfusion cannula inserted into

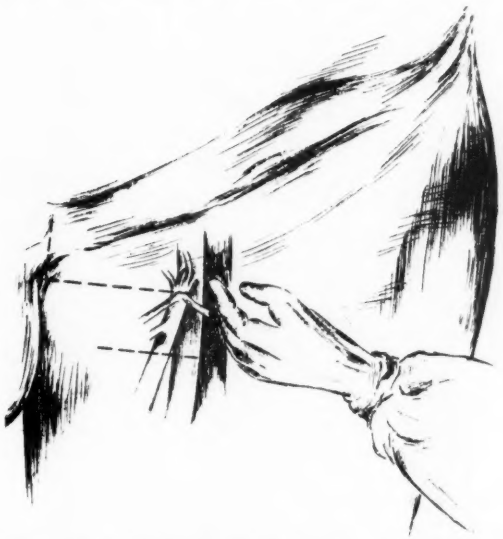

Fig. 16.—Anatomy—The femorosaphenous junction is usually 1.7 cm. below and 3.9 cm. lateral to lateral spine of symphysis. Incision 1 cm. below femorosaphenous point, horizontal and mesialward from femoral vessels. Landmarks: fingers on pulsating artery.

Fig. 17.—Local anesthesia.

the lumen of the vein. The two sutures are then tied, the lower one to prevent leakage around the cannula, and the upper to serve as a retractor on the upper stump, which is to be subsequently resected. The

vein is divided, and the field packed off for injection. The obturator of the cannula is removed, and a Luer-Lok syringe used to inject 2

Fig. 18.—Incision through the superficial fascia. The saphenous vein lies beneath the superficial fascia and on the fascia lata.

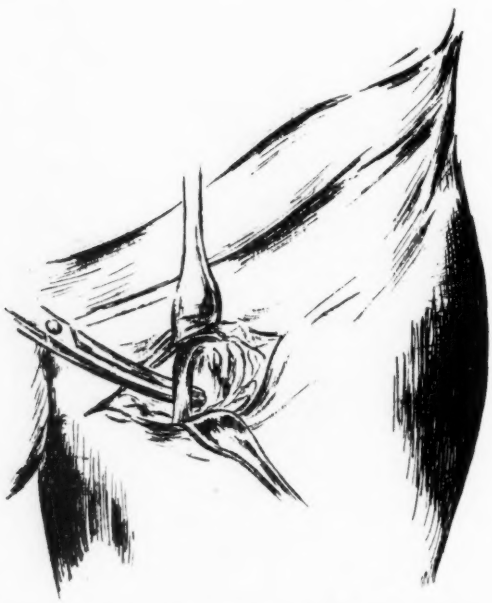

Fig. 19.—Blunt dissection is used to strip up saphenous vein.

to 5 c.c. of 5 per cent sodium morrhuate into the lower end. It is important to use a cannula rather than a needle for this injection, as insertion of a sharp needle into the lumen with the vein collapsed is

difficult. Having completed the injection, the obturator is replaced, the cannula removed, and the lower end of the vein again tied off.

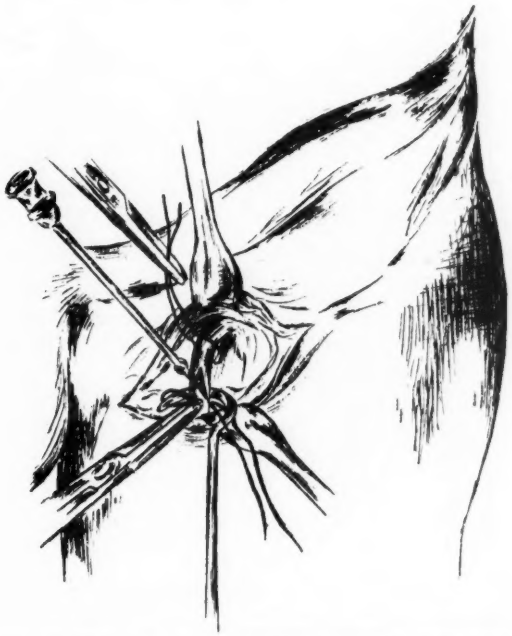

Fig. 20.—A nick cut in vein and a transfusion cannula tied into place. Vein tied off above and divided.

Fig. 21.—Distal portion of vein injected with 3 c.c. of sodium morrhuate, 5 per cent. Cannula removed, distal portion of vein religated and dropped out of site.

Then follows the most difficult part of the operation, the removal of the saphenous stump and division of the branches usually encountered at the femorosaphenous junction. This is somewhat tedious and

time consuming, but important. Using the upper ligature for traction, the stump is raised and freed by sharp dissection to its entrance into

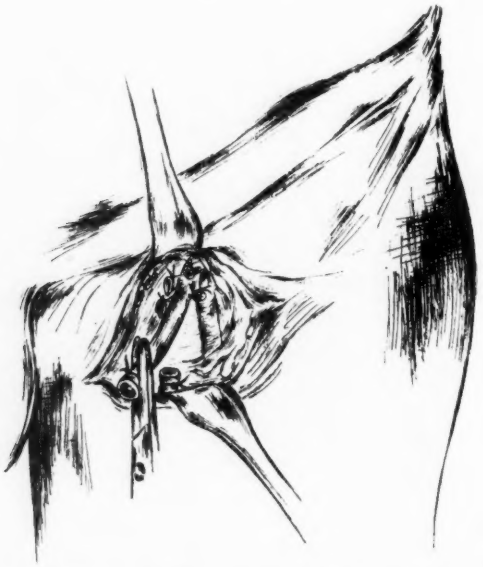

Fig. 22.—Wound retracted upwards, proximal end of vein dissected up to its entrance into femoral. All branches of saphenous and femoral at junction are ligated and divided.

Fig. 23.—The proximal end of vein ligated and divided flush with femoral vein. No stump allowed to remain.

the femoral. The tributaries at the junction are separately dissected and divided between ligatures. These tributaries may be several or all of the following: (1) a lateral superficial femoral vein; (2) a group

of medial superficial femoral veins; (3) a superficial circumflex iliac vein; (4) superficial inferior epigastric veins; and (5) a superficial external pudendal vein. The vein is then doubly ligated flush with the femoral and excised. If properly done, no stump is left that may gradually elongate into a recurring varix; no tributaries are left to dilate and reestablish downward flow; no collaterals are spared to varicose under the strain of increased load. The wound is closed in the usual manner. Interrupted deep sutures are placed to obliterate the dead space; continuous dermal is used for the skin. Occasionally, a small Penrose wick is placed in the wound for twenty-four hours. I arrived at the above described procedure independently, in a constant effort to improve my results in treating varicose veins. A review of

Fig. 24.—Special suture used to give good approximation of wound edges.

the literature early indicated to me how far from original my procedure was, other workers having encountered the same problems and solved them in the same way.

Opinions vary as to the advisability of doing the saphenous injection at the time of the ligation operation as described above. In my judgment, the simultaneous rather than subsequent injection is the procedure of choice. The chief disadvantage is the possibility of rendering the patient bed-ridden because of undue reaction in the injected vein. This is readily avoided by controlling the extent of the vein affected by the injection, by a secondary operation lower in the leg of which I shall say more shortly. The chief advantage of simultaneous injection is that the sclerosing agent is injected in a completely collapsed vein, with greater likelihood of permanent obliteration. Furthermore, the

uppermost portion of the varix is occluded by the injection, whereas this end is very difficult to inject at a future date.

The after-care of the combined ligation and injection procedure is simple. The patient is allowed to sit up in bed as soon as he desires and has lavatory privileges in three or four hours. The next morning, he leaves the hospital and resumes his usual activities. During his twenty-four hours in the hospital, large, warm, wet dressings of magnesium sulfate are applied to the injected saphenous to minimize reaction. Strips of elastoplast are placed across the vein in the thigh to support it during the next few days, and the patient wears an Ace bandage about the lower leg. Any veins not obliterated by the saphenous ligation and injection are injected at subsequent office visits. With the downward venous pressure removed, these injections are unusually effective and only a few treatments are required.

CONTROLLED INJECTION OF THE SAPHENOUS

Anyone who has used the injection method at all extensively has seen severe lymphedema follow an unexpectedly severe venous reaction. The lymphatics, in intimate association with the veins, are strangulated by the perivenous reaction. The resultant leg swelling may take weeks or months to disappear. I am well aware that some authors advise injection of all the varicosities at one sitting, and recall that Unger, of Leipzig, recommended inserting a ureteral catheter down the saphenous to below the knee and injecting the entire vein as the catheter is withdrawn. Certainly needless leg edema must be produced by such methods. I am sure these ideas are wrong. All the advantages of ligation and simultaneous injections are obtained without undue inflammation, and resultant edema, if the segment of vein injected is limited. To this purpose, I regularly ligate and divide the saphenous just above the knee, immediately before performing the ligation and injection operation in the groin. The gratifying result of a painless postoperative period without unexpected edema is well worth the extra effort.

Recurrences following combined ligation and injection procedures have been rare and usually confined to a few veins in the lower leg. From 1932 to July, 1936, forty-nine cases have been treated by the combined procedures outlined in this paper. The results over the four-year period have been as follows:

The three cases of recurrence had a doubly positive Trendelenburg test, indicating incompetency of the communicating veins.

FORTY-NINE CASES OF COMBINED LIGATION AND INJECTION

Cured	44	89%
Fair	2	4%
Recurred	3	7%

CONCLUSIONS

The injection treatment of varicose veins is adequate for relatively small and isolated varicosities. Recurrence is the almost invariable result, when this method is employed alone, for larger varicosities, directly related to the incompetent long and short saphenous vein. Such cases require interruption of the downward venous pressure at the so-called "blow-out" sites. Ligation of the long saphenous is performed ideally at the femorosaphenous junction, along with division of all tributaries encountered at this site. "Blow-out" sites lower in the leg require separate division below the lowest competent vein valve. In the intelligent use of the tourniquet following the principles of the Trendelenburg test, rests the key to the discovery of the various "blow-out" points. Undue inflammatory reaction following combined ligation and injection can be prevented by a second ligation, lower in the leg, to limit the extent of vein affected by the sclerosing solution. If the principles of combined ligation and injection as outlined in this paper are followed, the treatment of varicose veins should remain simple, ambulatory, and attended by a minimum of recurrence.

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SPONTANEOUS CLOSURE OF ARTERIOVENOUS FISTULAS

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THE spontaneous closure of small arteriovenous fistulas is not unusual, but little has been written on the subject. Discussions are to be found in Makins' book,[6] and in the studies of Reid,[8] who states that "some of the cases will heal spontaneously during the first six months, especially if the patient and the part are put at rest. Indeed, some arteriovenous aneurysms have been known to heal spontaneously after having been present for from five to eight years (Makins, Cushing[2])." The most remarkable case is that reported by Dry and Horton,[3] in which the fistula, produced by a bullet passing through the upper third of the right thigh, healed spontaneously after twenty-six years. All those who have done experimental work will agree with Brooks[1] that "even comparatively large fistulae established by careful vessel suture, in most instances close spontaneously."

Reid observed healing in Case 24 of his series. The patient was a white laborer, aged thirty-eight years, with a fistula in the right subclavian region, the result of a bullet wound. There was a very marked thrill and bruit, accompanied by rapid enlargement of the cervical veins. Operation was deferred, and after about two months the thrill and bruit began to disappear and the pulsation became less. At the end of six months the signs of an arteriovenous aneurysm had disappeared. In Reid's Case 25, a fistula occurred in the third portion of the left subclavian artery. The patient, a white laborer, aged forty-one years, had been shot six months previously. At first, the thrill was so marked that "the covers of the bed seemed to quiver." The intensity of the thrill decreased rapidly during the first month; after that it decreased more slowly. At operation, six months after the injury, the fistula was found to be small, and Reid noted that a spontaneous cure might have resulted if he had waited longer.

In Cushing's case (No. 5 in Reid's table), a carotid-cavernous sinus communication, with pulsating exophthalmos, had been present for three years. Cushing ligated the internal carotid artery, but it was only at the end of eight years that the symptoms and signs finally disappeared.

The experience of Makins may be summed up by quotations from his book: "We are well aware . . . . . that the general tendency is for these openings to contract in size, and even to close spontaneously. I have observed this latter result in its various stages in two instances

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of carotid arterio-venous aneurysm, in which primary consolidation of the sac was induced by proximal ligature of the common carotid artery. In both these cases the venous roar was reduced or disappeared after the operation, only to return to its original strength in a few days. In both it subsequently gradually decreased in strength, and after intervals of fifteen and twenty months respectively, finally disappeared. . . . . I have also had the opportunity of observing continuously the slow contraction and eventual complete closure of an arterio-venous communication between the innominate artery and vein, the process extending over a period of five years.'' He writes on another page: ''Clinical evidence is not wanting of early spontaneous closure of arterio-venous communications. Thus, in a man with a traversing bullet wound of the right thigh, ten days after the injury a local arterio-venous murmur was present in Scarpa's triangle, and a loud similar murmur was audible over the base of the heart. A fortnight later, both the local and the cardiac murmurs had completely disappeared, and no sign suggestive of any vascular injury remained.''

Plisson's[7] advice on small arteriovenous aneurysms follows: ''A primary point appears demonstrated: it is that certain small arteriovenous aneurysms are capable of healing spontaneously (Baudet). Therefore, if the patient does not complain of pain, if the aneurysm does not enlarge, does not threaten any complication, is not accompanied by any annoyance, it is better to abstain from operation. Pozzi observed a carotid-jugular aneurysm to heal thus spontaneously at the end of eight months.''*

Holman,[4] placing emphasis on large communications, sees danger in waiting for the spontaneous closure of some of the fistulas: ''If there is early evidence that the heart is increasing in size and that the blood pressure and pulse rate show marked variations on opening and closing a fistula, one may be certain that the lesion will not heal and must be eliminated by operation to avoid further effect upon the heart.'' Another indication for early operation is exemplified in my own case of arteriovenous aneurysm of the left brachial artery, due to a stab wound, in which, on the eleventh day following the injury, a sudden increase in extravasation of blood with consequent tension and pain in the arm necessitated ligaturing of the artery and accompanying veins. The ligatures were applied 2 cm. below the profunda brachii artery and circulatory disturbances did not result.

AUTHOR'S CASES OF SPONTANEOUS (NONOPERATIVE) CLOSURE

CASE 1.—A colored laborer, aged thirty-four years; stab wound, subclavian arteriovenous fistula; venous thrombosis; gradual reduction of venous pressure, increase in arterial pressure, cessation of thrill and diminution of bruit. The patient was stabbed below the middle of the left clavicle with a narrow knife, on Sept. 22,

*Author's translation.

1935. When admitted, his clothes were saturated with blood and the wound was oozing freely. He was in moderate shock and there was pain on motion of the left shoulder, arm and hand. The arm was cold and there was congestion of the veins, but no neurological changes. The clean-cut wound was closed with silk sutures and pressure was applied, but a large hematoma formed in the posterior axilla, which caused fever and disability.

On the tenth day, a thrill and loud to-and-fro murmur were noted over the swollen, pulsating region beneath the middle and outer thirds of the clavicle. The radial pulse was weaker on the left than on the right; the arterial pressure, left arm, 90/78; right, 145/85. The veins in the left arm were markedly dilated. On the next day, the murmur was not as loud as before, and neither the thrill nor the radial pulse could be felt. Two days later, a feeble pulse was made out on the left. The veins were distended and tortuous, and the patient complained of pain in the region of the shoulder joint. On the following day the pulse could not be felt, and the brachial vein and the veins of the cubital fossa were thrombosed and tender. The murmur continued in the left infraclavicular region. A day later, the arterial pressure in the left arm was 0, on the right, 140/60. The venous pressure on the left was 200 mm. (1 per cent sodium citrate), on the right, 90 mm. An electrocardiogram showed abnormality of the S-T segments, left axis deviation, and sinus tachycardia. The blood Kahn reaction was negative. Red blood count, 3,800,000; hemoglobin (Tallqvist), 75 per cent. For several days there was weakness of the muscles supplied by the median nerve.

On the twentieth day in the hospital, the left radial pulse was very weak and the arterial pressure on this side could not be obtained. On the twenty-third day, the pulse was stronger and the arterial pressure on the left was 112/90, on the right, 145/80. The venous pressure on the left was now 110 mm., on the right, 85 mm. On the twenty-eighth day, the arterial pressure on the left was 134/88, on the right, 155/88. The veins of the left arm were now less distended and the systolic bruit in the left infraclavicular fossa could barely be heard. On the day of discharge, Oct. 21, 1935, the venous pressures were approximately equal on the two sides, at 60 mm. I have not been able to follow this patient further, but there is little question but that the fistula has closed.

CASE 2.—An Irish farmer, aged twenty-eight years; shotgun wound, brachial arteriovenous fistula; visualization by neoskiadan; sudden, complete obliteration of the fistula within twenty-four hours after the injection. The record, made at the time of the second admission to the hospital, April 16, 1934, follows: "The patient is a tall, lean, farm laborer who was injured thirteen weeks ago by a shotgun from a distance of about 80 feet. He was admitted to our wards two days later. Small bullet holes were found scattered from the right wrist to the right shoulder, and there were several over the chest wall. There was moderate infection about most of them, and he was treated for ten days with hot packs. Several of the more superficial bullets were extracted. The patient now says that he felt a thrill over the lower, inner portion of his right arm before he left the hospital, but he did not tell the doctors about it. A roentgenogram taken at the time showed a flattened, irregular bullet at the top of the olecranon fossa, and this was assumed to account for the patient's inability to extend the forearm completely. Evidently no thorough examination of the vessels was made.

"In the interval since the first admission, the patient's wounds have all healed except for slight suppuration around one or two bullets on the medial side of the arm. But, for the past four weeks he has had considerable aching pain in the right forefinger, which spreads at times into his thumb and middle finger. He now milks only with his left hand. The right arm feels heavy and there has been aching above the elbow.

"Examination shows a thrill and a continuous machine-like murmur with systolic accentuation, loudest just medial to the biceps muscle in the lower third of the right arm. It is heard anywhere over the arm, but is not audible when the stethoscope is placed over the right subclavian artery or over the heart. The veins of the arm and forearm are moderately dilated. The fistula is easily obliterated by light direct pressure, and the thrill and bruit disappear when the artery is compressed against the humerus proximally.

"The radial pulse on the right is only slightly weaker than on the left, and the circulation in the forearm and hand at first sight appears good. When the hand is elevated, however, it becomes markedly pale, and when it is depressed it flushes. If the brachial artery is compressed above the level of the fistula without obliterating the communication, the arm and hand become ivory white. The arterial pressure in the affected arm is 120/76, and in the opposite, 110/68. After obliterating the fistula the arterial pressure in the other arm is unaltered (108/68). When the cuff is put on the left forearm, the oscillations due to the arterial pressure are seen easily in the mercury column, but when the cuff is put on the right forearm there are no oscillations. Three pulse readings, at three-minute intervals, were 79, 76, and 76 beats per minute. Pulse rates, taken alternately with these, during times when the fistula was closed off, were 68, 64, and 66, demonstrating a slight, but definite, Branham bradycardiac phenomenon.

"Light compression on the arm above the fistula causes tense filling of the cubital veins, and aspiration of blood from these veins shows admixture with arterial blood."

A determination of cardiac output was made by John Walker Moore and J. Murray Kinsman, who used their dye injection method.[5] The curve was diphasic, indicating by the second rise the effect of the shunting of part of the dye through the fistula. The output in liters per minute (corrected for body surface area) was 5.28. The average normal is 3.79, and the normal for this particular patient, determined later, was 3.65. The increase in output, due presumably to the presence of the fistula, was therefore, 44.7 per cent.

An arteriogram was made by the following method: A tourniquet was applied tightly to the upper arm, and 15 c.c. of neoskiadan were injected into the cubital vein. Roentgenograms, with the needle in place, were taken immediately and were reported upon by S. E. Johnson: "Reexamination of the right elbow . . . shows marked dilatation of the lower third of the basilic vein. The vena comites and brachial artery are well filled from the upper limit of the film downward to its bifurcation. The exact level of the arteriovenous fistula cannot be definitely established."

On the morning after the injection of neoskiadan, the thrill and bruit had disappeared completely. There was no evidence of thrombophlebitis. When seen eight and one-half months later, Jan. 18, 1936, a few of the shot were still palpable, movements were normal at the elbow, there was no evidence of fistula, and the radial pulses were equal in volume.

A second cardiac output determination made thirteen months after obliteration of the fistula showed a monophasic curve and a reduction in output to within the normal range. Here is a comparison of output values before and after obliteration:

	TYPE OF CURVE	CORRECTED OUTPUT (LITERS PER MINUTE)	WORK (KG. METERS PER MINUTE)
Before	Diphasic	5.28	13,875
After	Monophasic	3.65	9,820
Average normal	Monophasic	3.79	4,690

CASE 3.—A colored male, aged thirty-four years; stab wound, brachial arteriovenous fistula; intermittent mechanical compression, followed three months later by cessation of pulsation and thrill; bruit absent on subsequent examination. The patient was admitted to the hospital on April 27, 1935, suffering from shock, the result of two recently inflicted stab wounds, one in the upper left flank and the other on the inner side of the right arm just below the axilla. The wound in the flank was found to be superficial, and both were debrided and closed without drainage. Ecchymosis and diffuse swelling persisted about the upper arm for two weeks, and when this subsided there was noted a more circumscribed, pulsating mass, about three centimeters in diameter, over which a thrill was felt. On applying a stethoscope there was "a constant roar, intensified by a pounding noise, synchronous with each heart beat." The arterial pressure in the unaffected arm was 136/88, but on attempting to read it on the right, no clear-cut sounds could be heard; the roar was continuous from 0 up to 300 mm. and over. The radial pulse volume was very small on the right and by palpation the beats could barely be felt at about 68 mm. The heart-chest ratio in a roentgenogram, exposed at six feet, was 10 to 31 cm. A roentgenkymogram showed greatly diminished excursion of the left ventricle and aorta. An electrocardiogram indicated sinus tachycardia and left axis deviation. The red blood count was 3,730,000; hemoglobin (Tallqvist) 80 per cent. The blood Wassermann reaction was negative. The cardiac output, determined by Moore and Kinsman, was 5.32 liters per minute (corrected for body surface area).

The following note was made eleven months later (March 4, 1936): "When the patient left the ward he was instructed to compress the arteriovenous aneurysm. Each night he lay on his right side and obliterated it completely with the palm of his left hand for half an hour or more at a time. Sometimes he would strap a pad against the mass. He continued to feel the pulsation and thrill for about three months, when both disappeared. None of the signs have recurred. The patient had a complete wrist-drop when he left the hospital, and was unable to use his forearm or any of the muscles in his hand. After about five months, he was able to dorsiflex the wrist, and some of the other motions of the hand have returned.

"Examination now shows a scar one centimeter long on the medial aspect of the right arm, between the biceps and triceps, just below the insertions of the pectoralis major and latissimus dorsi muscles. There is a hard, nontender, fusiform mass in the brachial artery at this level. The artery pulsates strongly above the mass, but below it there is no pulsation. There is no thrill or bruit. The brachial artery cannot be felt in the lower third of the arm. The radial artery is palpable at the wrist, but the impulse is much weaker than on the other side. The arterial pressure in the unaffected arm, where the oscillations begin at 170 and stop at 30, is 140/84. On the right, no arterial sounds can be heard, but oscillations are seen between 160 and 50. Examination of the forearm and hand shows loss of function of the supinator longus, the opponens pollicis, and the first and second lumbrical muscles. There is radial and ulnar weakness as well, with moderate atrophy of the hypothenar eminence, and marked atrophy of the thenar eminence. There are no sensory changes to pin-prick."

CONCLUSIONS

1. Small traumatic arteriovenous fistulas often heal spontaneously.

2. Closure appears to be accelerated by spontaneous venous thrombosis or by systematically repeated obliteration by compression.

3. The possibility of the use of sclerosing solutions suggests itself and may perhaps find limited application when damage to the intima can be confined to the fistula and the vessel walls immediately adja-

cent to it. Injection treatment has been used successfully in two small congenital fistulas by Smith and Horton.[9]

4. Fistulas, even when small, increase the cardiac output.

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SUBCUTANEOUS EMPHYSEMA ASSOCIATED WITH PERFORATED PEPTIC ULCER

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SUBCUTANEOUS emphysema arising from injury or disease of the chest and respiratory passages is of frequent occurrence on a busy surgical service, but emphysema arising from some solution of the continuity of the gastrointestinal tract is an unusual finding. One of the most interesting sources of subcutaneous emphysema of intestinal origin is that which is seen rarely in association with perforated ulcer of the stomach or duodenum. The physical examination, roentgen ray films, and operative findings in this case serve to illustrate this striking phenomenon.

CASE REPORT.—F. K., CGH68429, aged sixty-two years, male, married, white. The patient entered the hospital on Feb. 12, 1937, and gave the history that two days previously while at work he was seized with severe cramplike abdominal pains which seemed to arise in the left upper quadrant of the abdomen but which quickly spread over the entire abdomen without radiation to other parts. On the day before entry the cramps were less severe and he vomited twice some medication that was given to him. On the day of his entry he complained of very little abdominal pain. About one week before entry the patient began to notice an occasional cramplike midabdominal pain and became more constipated than usual. Five days before entry he took a cathartic and had a bowel movement the following day, but he was obstinately constipated thereafter in spite of enemas and medication. There was no history of melena, but he had had some bleeding hemorrhoids many years ago. On the evening preceding his entry into the hospital, his family noticed a slight swelling of the left side of the neck. On the morning of Feb. 12, 1937, while straining at stool attempting to evacuate an enema, a diffuse swelling of the neck occurred rather suddenly. The abdomen had gradually become distended over the previous two days, following the onset of his episode of acute abdominal pain. He had had a cold with a rhinitis, cough, and a small amount of purulent sputum over the previous six weeks. He had a chancre in his youth. Over the past year he had lost ten pounds in weight. There was no history of any sort of digestive disturbance.

Physical examination revealed a thin, pale, sallow white male of about sixty years of age who was in no acute pain or distress. The head was partially bald. Pupils almost completely fixed. Nasal septum intact. Pharynx mildly injected. There were a few dental snags. There was an extensive subcutaneous emphysema of the entire neck and this extended downward over the anterior aspect of the chest, both axillae and to some degree on both upper arms. There was no respiratory embarrassment. Breath and voice sounds were somewhat distant and both diaphragms were high. The trachea was in the midline. The heart sounds were of fairly good

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quality, rhythm regular, no murmurs present. Vessels somewhat sclerotic; B. P. 176/110. The abdomen was greatly distended and tympanitic, and felt like the pneumoperitoneum that is induced in peritoneoscopy. There was only slight tenderness throughout with no rebound tenderness and no muscle guarding. Peristaltic activity was diminished and heard as though at a distance. The area of liver dullness was almost completely obliterated by tympany. No evidence of increased intra-abdominal fluid could be elicited. Genitalia normal male. Rectal examination revealed the presence of hemorrhoids; the prostate was small; high in the culdesac acute tenderness referred to the lower abdomen was obtained on palpation. Extremities thin. Left knee jerk absent. Right Achilles reflex greater than the left. Other reflexes hyperactive. Temperature, 98° F. White blood cell count, 4,200. Urinalysis revealed only the presence of an occasional pus cell.

A tentative diagnosis of the perforation of a hollow intraabdominal viscus with pneumoperitoneum and subcutaneous emphysema was made. In view of his con-

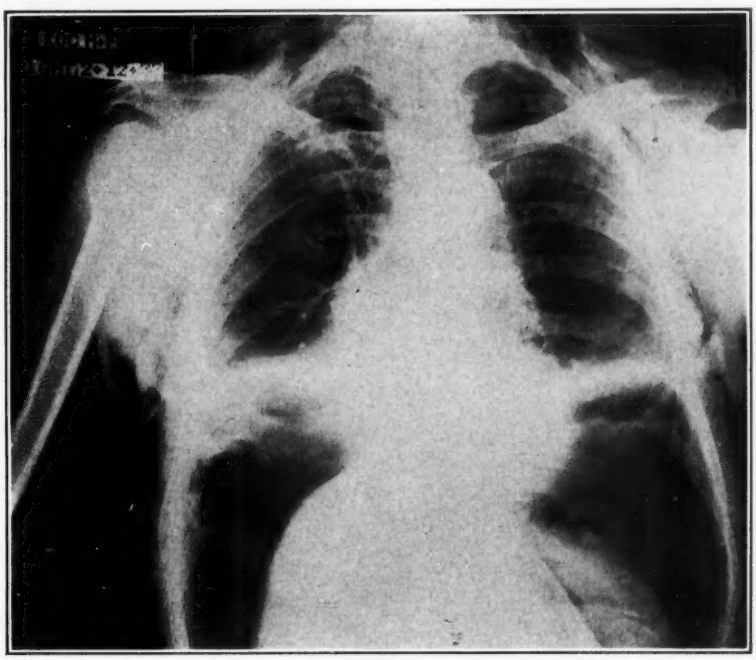

Fig. 1.—Roentgenogram showing the large accumulation of gas beneath the diaphragms and in the tissue of the neck and axillae.

stipation, loss of weight, and cramplike pains, it was thought most likely that he had a perforation of a malignant ulcerative lesion of the colon, or a perforation of the cecum secondary to a malignant occlusion of the colon. Perforation of the duodenum, however, was also kept in mind.

Roentgen ray examination of the abdomen revealed an extensive pneumoperitoneum. In the chest plate the diaphragms were displaced upward and the lung bases were somewhat compressed as a result of the pneumoperitoneum. There was a small amount of gas in the intestines. There was an extensive emphysema of the mediastinum, neck, and chest wall. A barium enema entered the large intestine freely to a point about six inches proximal to the splenic flexure, but no evidence of annular deformity could be demonstrated.

Laparotomy was performed through a right rectus incision under local anesthesia supplemented by ether. As the peritoneum was opened, a large amount of gas

escaped with a hissing noise and the abdominal distention collapsed. There was a small amount of turbid (dishwater) fluid and an early generalized peritonitis. A small amount of fibrin was present in the right upper quadrant around the duodenum and gallbladder. On the anterior wall of the duodenum there was a small "punched out" (acute) perforated ulcer. No lesion was felt on the posterior duodenal wall. There was no demonstrable emphysema in the ligamentous structures or retroperitoneal region as far as exploration was possible. The ulcer was cauterized and closed with a miniature Heineke-Mikulicz pyloroplasty. The abdomen was closed with silver wire without drainage.

The postoperative course was stormy. The subcutaneous emphysema gradually subsided. He developed the signs of a progressive generalized peritonitis with

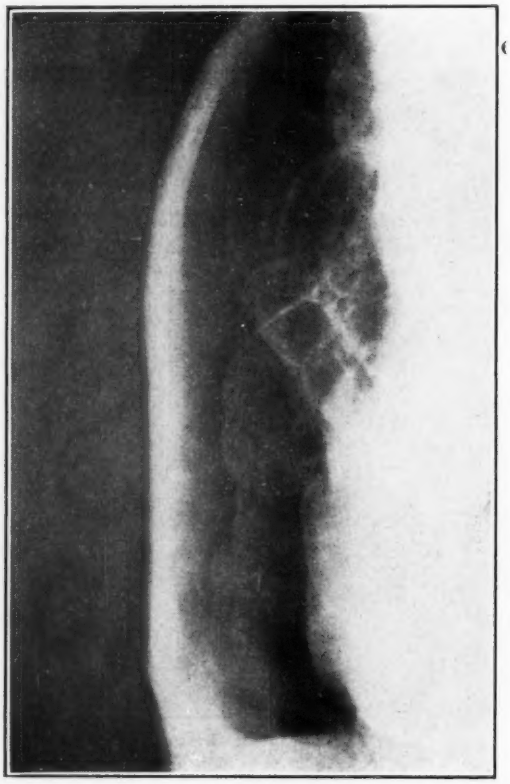

Fig. 2.—Lateral roentgenogram of the abdomen showing the extensive pneumoperitoneum.

fever, acute abdominal tenderness, and distention. The wound became infected and he developed a terminal bronchopneumonia and expired on the eighth postoperative day. Autopsy was refused.

Demarquay[3, 5] is said to have been the first to describe subcutaneous emphysema after the perforation of the gastrointestinal tract when the parietal peritoneum is injured.

Roger's[3] work, "De l'emphyseme generalise," was prepared on the basis of a case of very widespread subcutaneous emphysema following the perforation of a tuberculous ulcer.

Poensgen[3] gave his inaugural address in 1879 on the subject "Das subkutane Emphysem nach Kontinuitatstrennung des Digestionstraktus, inbesondere des Magens." In his discussion of this treatise Korach[3] states that Poensgen collected illustrative cases, went into the subject exhaustively, and reported a case studied in conjunction with the internist Kussmaul and the pathologist Recklinghausen. The case was one of perforation near the cardia with an extension that spread the muscular fibers of the esophagus. Kussmaul believed that the gas got into the mediastinum through the split fibers of the esophagus, and from the mediastinum finally found its way into the subcutaneous tissues. Recklinghausen held that the gas progressed subperitoneally from the ulcer to the region of the diaphragm from where it went to the anterior mediastinum or to the retroperitoneal cellular tissues and thence found its way to subcutaneous tissue.

Faber, 1885, is quoted by Hurst and Stewart[1] under their discussion of "gastromediastinal fistula." Faber's case was one in which an ulcer of the lesser curvature perforated directly into the mediastinum with resulting mediastinal and subcutaneous emphysema.

Korach[3] states that this symptom is mentioned in large books on the diseases of the stomach, e.g., Boas, Riegel. It is also spoken of in certain collected essays, e.g., Brütt, Mühlsam.

Vigyázó[7, 8, 9] in 1926 reported the case of a fifty-two-year-old tabetic in which the diagnosis between peritonitis and a tabetic gastric crisis was difficult. He saw the patient about one-half hour after onset of pain. The discovery of a subcutaneous emphysema about the region of the umbilicus on the abdominal wall aided him in making the diagnosis. At laparotomy he found a pinhead sized perforation of a calloused ulcer of the anterior wall of the duodenum. The emphysema disappeared on the first postoperative day. He has two theories advanced to attempt to explain the condition. First is that the gas might diffuse through the parietal peritoneum. Second is that the gas finds its way subserously either along (a) the hepatoduodenal and round ligaments to the umbilical region, or (b) subserously to the retroperitoneal tissues and then around the flanks to the abdominal wall anteriorly. The first variation of the second theory he believes to be the most likely and the valvelike appearance at the opening of the perforation in his case would tend to substantiate it. He believes the reason for the infrequent occurrence of the condition is the fact that the mechanical conditions at the site of perforation are not favorable for the diffusion of gas in the manner described.

Podlaha[6] in 1926 reported the case of a patient, aged seventy-three years, seen twenty-six hours postperforation who had a greatly distended tympanitic abdomen and subcutaneous emphysema of the left supraclavicular fossa, the jugular notch, and the anterior thoracic wall. At laparotomy he was found to have a perforated ulcer high on the

lesser curvature near the cardia of the stomach. His emphysema subsided over a period of five days. He expired on the fourteenth day with a left subphrenic abscess. Podlaha states that the perforation is usually located so that the gaseous contents of the stomach escape into the peritoneal cavity. If, however, the perforation occurs high on the lesser curvature, or on the posterior duodenal wall near the insertion of the hepatoduodenal ligament, and if the perforation is small with a flap valve or irregular wall, the gas can get into the interstitial tissues and find its way to the subcutaneous regions. In the case of the duodenal ulcer it may go to the region of the umbilicus and in those ulcers near the cardia it goes to the left supraclavicular fossa. Experimentally in ten cadavers and in three dogs, Podlaha demonstrated the methods of diffusion of gases from the various parts of the stomach and duodenal regions by injecting hydrogen peroxide into the subserous tissues. He found that from injections made in the region of the pylorus the material passed along the hepatoduodenal and round ligaments (of the liver) to the region of the umbilicus. From injections made near the cardia, the material made its way principally by way of the phrenicoesophageal ligament and aortic hiatus into the posterior mediastinum, through the cleavage planes to the neck, and to the subcutaneous tissues. Podlaha gives three possible reasons for the rarity of subcutaneous emphysema with perforated ulcer:

1. Perforations on the cardia and posterior wall of the duodenum are rare.

2. The perforation must be small and in the form of a zig-zag fissure or valvelike.

3. The interstitial connective tissue must not contain too much fat.

Korach[3, 4] in 1927 reported the case of a thirty-six-year-old man who was seen about eight hours postperforation with a large pneumoperitoneum and a small area of emphysema about the region of the umbilicus. The patient appeared to be dying at that time. Korach saw the cadaver a short time later and the emphysema had spread over the face, neck, left orbit, chest wall, and over the anterior abdominal wall to the flanks. At postmortem examination a large amount of gas escaped from the peritoneal cavity with a hissing sound. This gas exploded, when ignited, with a blue flame. The gas from the subcutaneous areas behaved in a similar manner. On the visceral and parietal peritoneum in several places were dark necrotic patches. There was an emphysema in the peritoneal leaves of the mesentery, colon, ileum, and root of the mesentery. The stomach was dilated and the pylorus almost occluded. High on the lesser curvature near the cardia was a large discolored area containing at its center a circular, smooth-walled perforation. There were about three liters of dark fluid in the abdomen containing food particles, principally partially digested bread and potato. He emphasizes that

this was not a phlegmonous emphysema. He calls attention to the particular interest of the emphysema spreading after death and to the identification of the gas as hydrogen. He gives a presumptive formula for the formation of hydrogen gas from the dextrose of the partially digested food in the stomach. He hypothesizes also that the large amount of gas generated in the partially occluded stomach might have been responsible for the perforation. From the study of his case, Korach believed that the gas diffused directly through the peritoneum where its structure was altered or destroyed by the inflammatory process.

Kellogg[2] in his monograph *The Duodenum*, 1933, deals very briefly with emphysema: "Emphysema of the surface of the body, which is infrequent, suggests perforation of the digestive tract or an injury involving the respiratory tract."

Examination of those recent texts and systems of surgery at our disposal reveals no mention of this unusual phenomenon.

From this brief survey of the literature it seems that the explanation of the appearance of subcutaneous emphysema in perforated ulcer of the stomach or duodenum is not clear. Certainly in our case the route followed by the gas from the stomach to mediastinum and the subcutaneous tissues is not obvious.

Two theories as to the possible routes of diffusion have been advanced:

1. Diffusion directly through the parietal peritoneum. This would seem to be particularly feasible in cases where the pneumoperitoneum was extensive and under pressure (as in our case) and where the parietal peritoneum was greatly damaged by the inflammatory process (as in Korach's case).

2. The diffusion of the gas from the site of perforation subserously (a) along the peritoneal covered ligamentous structures about the stomach and duodenum and then to the neck by way of the mediastinum or via the round ligament (of the liver) to the umbilical region; (b) to the retroperitoneal areolar tissues and then around the abdominal wall or up the mediastinum to the neck. The cases of Podlaha and Vigyázó tend to support the first section of this theory as do the experiments of Podlaha on the injection of hydrogen peroxide subserously about the stomach and duodenum in cadavers and in dogs. This theory requires, however, that certain mechanical conditions be present at the site of perforation; viz., a valvelike flap or an irregular linear perforation that would permit the "pumping" of the gas into the subserous tissues from the stomach.

Vigyázó[9] and Korach[3] brought up the possibility of a ruptured pulmonary emphysematous bleb into the mediastinum as the origin of emphysema in Podlaha's case. The same criticism might be applied to our case. Although there was no roentgen ray or physical evidence of

pulmonary disease at the time of preliminary examination, we are unable to offer the final proof because necropsy was refused.

This association of emphysema with perforated ulcer does not lend itself to the same easy explanation that can be advanced for the emphysema that is sometimes reported following operative procedures on the intestinal tract or penetrating injuries of the abdomen because in these situations the parietal peritoneum is broken.

It must be said that the phenomenon of subcutaneous emphysema seen with perforated ulcer should not be confused with the phlegmonous gaseous infiltrations that sometimes occur following the retroperitoneal laceration of the duodenum, colon or rectum.

Korach's studies on the hydrogen content of the gas from the peritoneal cavity and emphysematous tissues in his case are interesting but inconclusive because they were done postmortem.

SUMMARY

1. A case of perforated duodenal ulcer associated with an extensive pneumoperitoneum and a subcutaneous emphysema of the entire neck, chest wall, axillae, and upper arms is reported.

2. The available literature regarding similar cases is reviewed briefly.

CONCLUSION

A completely satisfactory explanation of the occurrence of subcutaneous emphysema in rare cases of perforated stomach or duodenal ulcer is not available at the present writing.

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THE USE OF ZINC PEROXIDE IN MICROAEROPHILIC INFECTIONS

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ZINC PEROXIDE was introduced for surgical use by Meleney[1] in 1935. Its peculiar value lies in its ability to liberate oxygen over a period of many hours which renders it nearly specific in certain anaerobic infections. The fact that it is practically devoid of irritant properties is an additional advantage of great importance.

In 1935 Meleney[1] reported a group of cases of spreading ulceration of the abdominal wall associated with a hemolytic microaerophilic streptococcus. No anaerobic cultures were made from the first case in the group which was included on the basis of clinical similarity to the remaining five. In all of these hemolytic streptococci were found on anaerobic culture. In two cases the organism also grew under aerobic conditions.

These infections usually start from a wound, either operative or traumatic, and run a very chronic course. Moderate fever is frequent. Pain may be severe but is inconstant. The lesion presents a dirty sloughing base. It slowly undermines the surrounding skin, breaking through to form sinuses opening a short distance from the margin of the original ulcer. These openings gradually enlarge to form secondary ulcers, which finally spread and coalesce with the primary one.

The lesions have been extremely refractory to the usual methods of treatment. A wide variety of antiseptics have been used without success. Repeated excision has, as a rule, been unavailing. Roentgen therapy has been tried without success. In the latter three cases of Meleney's series, zinc peroxide was applied with prompt improvement and subsequent complete healing. Later Meleney[2, 3, 4] reported the use of zinc peroxide in a variety of lesions due to anaerobic and microaerophilic organisms.

Zinc peroxide is a fine powder which is not available in pure form. Commercial brands contain 40 to 50 per cent of the peroxide, the remainder being composed of the hydroxide and the oxide. There is a great difference in the effectiveness of the products of various manufacturers which appears to be due not to the purity of the chemical but to an obscure difference in the availability of the oxygen.

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A simple test suggested by Meleney[4] for determining whether a given lot is effective consists in shaking a few grams of the powder in a test tube filled with water and allowing it to stand. The effective zinc peroxide forms a suspension which precipitates rapidly, leaving the supernatant liquid clear, and forms visible bubbles of oxygen in the precipitate within two or three hours.

On the basis of the information at hand, the product of the DuPont Chemical Company, of Niagara Falls, New York, prepared for medicinal

Fig. 1.—R. K. Leg ulcer due to microaerophilic hemolytic streptococci at the time of admission to the hospital.

purposes is the most reliable. The manufacturers advise heating the powder to 140° C. for four hours shortly before it is used.

It is applied in the form of a thin paste directly to the ulcer and should be covered with wax paper or gauze impregnated with zinc oxide ointment to prevent drying. Usually it forms a thin cake moulded by the walls of the ulcer. This should be removed once a day and replaced with a freshly prepared paste.

It has been the practice on Dr. I. S. Ravdin's service at the Hospital of the University of Pennsylvania to debride the ulcer just before be-

ginning the treatment and to facilitate epithelization with pinch grafts soon after the ulcer has become clean and started to granulate.

During the past two years, two patients with chronic leg ulcers infected with microaerophilic hemolytic streptococci have been admitted to the Hospital of the University of Pennsylvania. Since they demonstrate the great value of zinc peroxide, the salient points of their histories have been included.

CASE 1.—The patient (R. K.) was a twenty-two-year-old man who developed an indurated infected area over the left tibia in March, 1936. This was incised early in April. After it had drained for three weeks without tending to heal, the patient

Fig. 2.—R. K. End-result of ulcer shown in Fig. 1 after treatment with zinc peroxide.

came to the Surgical Out-Patient Department of the Hospital of the University of Pennsylvania, on April 27, 1936. At that time there were two shallow punched-out ulcers about ¾ of an inch in diameter, situated close together over the left tibia. These were treated with gelatine boots for one month with complete healing of one ulcer but not of the other. This remained open and in the course of a few weeks extended and the skin in an adjacent area broke down.

The area was drained on May 28, 1936, and for two weeks thereafter clean granulation tissue formed at the base. There was, however, a marked cellulitis about the ulcer which persisted. Three x-ray treatments were given with transient improvement. On June 19 another ulcer formed 0.5 cm. below the old one. On June 26 it was noted that the adjacent skin was being undermined, and this area was excised. During the next few weeks the ulcers coalesced to form an area 6.5 cm. by 3.5 cm. with a few small sinuses opening nearby.

On July 27, 1936, the patient was admitted to the surgical wards. At that time the ulcer had the appearance shown in Fig. 1 and measured 8 cm. in length and 3.5 cm. in width. X-ray of the tibia and fibula was negative.

Pus from the ulcer was cultured and showed hemolytic streptococci in pure culture both aerobically and anaerobically. On Aug. 3 zinc peroxide paste was applied. A fresh paste was prepared each day and the ulcer covered with it. Improvement was noted within three days.

By Aug. 13 nearly the whole area was epithelized. The remainder was clean and pinch grafts were applied. A number of these grew and epithelization was complete on Aug. 20. The patient was last seen in April, 1937. The ulcer has remained healed and painless (Fig. 2).

Fig. 3.—L. H. Leg ulcer due to microaerophilic hemolytic streptococci. This ulcer started over three months earlier from a minor abrasion.

CASE 2.—L. H., a white woman, thirty-seven years of age, was admitted to the Hospital of the University of Pennsylvania on March 4, 1937, with an ulcer 5.5 cm. long and 3.5 cm. wide on the right leg. It was situated over the subcutaneous portion of the tibia about 5 inches below the knee.

The lesion started from a slight abrasion caused by bumping a chair Dec. 16, 1936. At first there was considerable swelling and redness. Drainage started at the end of a week. After Jan. 1 the patient stayed in bed and the swelling subsided. Treatment consisted in salves, hot wet dressings, and Scharlach R.

At the time of admission to the hospital the ulcer was 0.5 cm. deep and irregularly oval in shape (Fig. 3). The edges were ragged and the bottom was covered with unhealthy granulation tissue covered with pus.

X-ray examination showed no underlying bone pathology.

A culture was taken and the unhealthy granulations and a portion of the edge of the ulcer excised and studied histologically. The culture showed hemolytic streptococci both aerobically and anaerobically. The pathologist reported the excised tissue as a nonspecific ulcerative granuloma.

The ulcers were covered with a thin paste of zinc peroxide which was renewed daily. Clean granulations grew up from the bottom. On March 11, 1937, seven Staige-Davis grafts were taken from the thigh and placed on these granulations. All the grafts took and the wound was about half covered with epithelium at the time the patient left the hospital on March 18.

Five days later the whole area was covered except for a spot 8 mm. in diameter. Healing was complete on April 2, 1937, and the patient has remained well (Fig. 4).

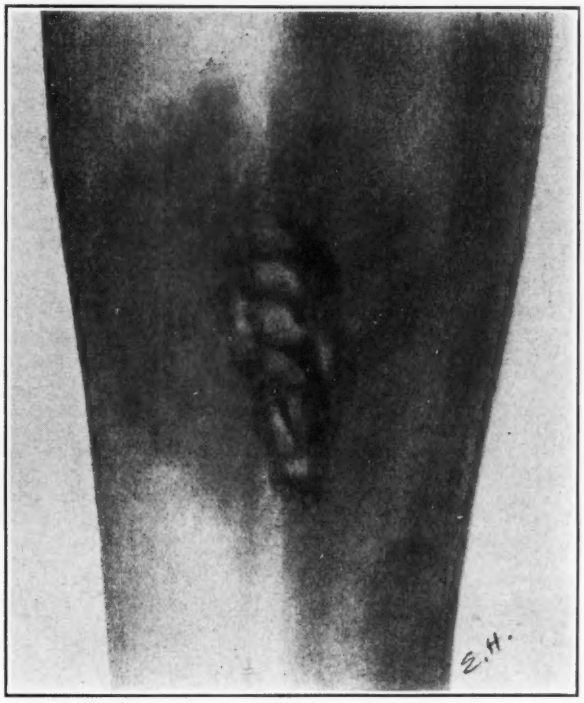

Fig. 4.—L. H. Ulcer shown in Fig. 3 after treatment with zinc peroxide.

In neither of these cases was the organism an obligate anaerobe. The ulcers were not on the abdomen and the patients were nearly afebrile. Otherwise, they correspond very closely to the original cases described by Meleney. While a majority of the cultures obtained by Meleney grew only under anaerobic conditions, two of them also grew in the presence of air as these did. In later reports Meleney[2, 3, 4] has described ulcers in locations other than the abdomen which have apparently had the same etiology as those in his earlier paper and have responded to the same treatment.

Recently a patient presented himself for treatment of a chronic high perirectal abscess which began just below the promontory of the sacrum

and from which a nonhemolytic streptococcus was cultured by anaerobic methods. The abscess opened into the rectum about 4 inches above the anus.

In this case zinc peroxide was diluted to the consistency of whitewash and administered daily as a low retention enema in amounts of 3 to 6 ounces.

CONCLUSIONS

Two typical cases of chronic undermining ulcer due to microaerophilic hemolytic streptococci are presented. Both healed rapidly after the use of zinc peroxide.

Zinc peroxide may be introduced into the large bowel with impunity and has proved useful in a case of perirectal abscess associated with a nonhemolytic streptococcus which grew anaerobically.

In the infrequent but refractory ulcers described above, zinc peroxide is nearly specific. As no other form of treatment has proved satisfactory in these cases, it is a needed addition to the surgeon's pharmacopoeia.

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

RECENT ADVANCES IN THE TREATMENT OF VARICOSE VEINS*

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INTRODUCTION

THE following discussion is confined to varices of the lower extremities. Only that treatment which deals with the varicosities themselves will be discussed. No attempt will be made to discuss the treatment of varicose ulcers other than the removal of the cause. Thrombophlebitis or other allied diseases of the venous system of the lower extremity will not be considered.

No attempt is made in this paper to cover the entire development of the treatment of varicosities. The general historical development is outlined and the more important phases of modern therapy are emphasized.

There has never been any wholly satisfactory treatment for varicose veins of the legs. This fact is emphasized by the numerous different treatments or modifications of treatments advocated and by the enthusiasm with which new treatments or new modifications have been seized upon. In all types of therapy tried there has been a relatively large percentage of recurrences in cases followed any length of time, in spite of the fact that the immediate results of most types of treatment have usually been encouraging. It seems the most logical reason for this state of affairs is that many types of therapy have been devised without taking into consideration the underlying pathology of various veins.

No types of therapy carried out for the relief of varicose veins of the lower extremities are new at the present time. All have been tried out before, have been discarded, and have reappeared. Their reappearance, however, has been marked by a distinct increase in the knowledge of the fundamental factors concerned and with more knowledge of the proper selection of cases, as well as purely technical improvements.

HISTORY OF THE TREATMENT OF VARICOSE VEINS

The recognition and the treatment of varicose veins of the lower extremities is as old as medical literature. Hippocrates[43] described a method of obliteration of a varix by passing needles into the veins. It

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is interesting to note that this first recorded attempt to treat varicose veins was an attempt to obliterate them in situ and is of course exactly the same in theory as the modern so-called injection treatment of varicose veins.

Mayo,[72] in speaking of the history of the treatment of varicose veins, mentions puncture with the Paquelin cautery or with the electrocautery, cutaneous and subcutaneous suture and ligature as practiced by Valpeau and Delpech. These were also methods of obliterating veins in situ which depended upon thrombosis followed by fibrosis.

The first surgical interference that is referred to in the literature is the ligation of a varicose vein above an ulcer, performed by Celsus[14] in the first century. This has also been referred to by Edwards,[33] who further states that the same procedure was carried out for the same purpose by Paré in 1579 and by Howe in 1797. De Takats[30] states that Howe advocated ligation of veins above ulcers and that his results were excellent except for the sepsis following operation. Brodie[10] in 1846 emphasized the rationale of ligation, especially the saphenous, but also recommended other methods of blocking the vein, such as by compresses or tampons (also referred to by Waimack[103]). In 1891 Trendelenburg[100] advocated ligating the saphenous vein above the knee for the relief of symptoms due to varicose veins.

Since that time the operation has been recommended by numerous other surgeons, most recently by De Takats,[27, 29, 30] in conjunction with injection. Edwards[33] gives an extremely interesting chart showing the approximate site of ligation practiced by different men. It is interesting to note that with few exceptions the more modern the surgeon, the higher the site of ligation. As has been stated, ligation alone of the saphenous vein or of one of its tributaries has never proved a wholly satisfactory procedure. Lower ligation merely shut off the backflow of blood above one certain point, usually an ulcer, and therefore improved the circulation at that point. The extremity as a whole, however, still retained varicosities. Although the improvement was often sufficient to heal the ulcer, collateral circulation would usually form around the obstruction and the condition would revert to its original status. With the development of surgery in general and especially with the decrease in danger from infection, more complicated operations were devised.

Schede[88] in 1877 described his operation, which consisted of a more or less extensive division between ligatures of the superficial veins above an ulcer. In 1884 Madelung[63] published the method with which his name is associated. This consisted in the complete extirpation of the entire greater saphenous vein and of as many of its branches as seemed indicated.

Thus, for a time, there were three well-recognized operations for varicose veins: those of Trendelenburg, Schede, and Madelung. Gen-

erally speaking, the two former operations seem to have given rather disappointing results, and the later operation was of greater magnitude than many cared to undertake. Miller[74] in 1906 reviewed the results of these three operations as carried out at the Johns Hopkins Hospital. He stated that the Trendelenburg operation had been carried out on nineteen patients, and the Madelung on only five. He further stated that these five cases were all from among the early admissions to the hospital and could not, therefore, be traced. He thought this unfortunate, since he considered that probably this type of operation was followed less frequently by recurrence than either the Trendelenburg or the Schede operation.

In 1899 Casati, referred to by Miller,[74] described a method of tearing out the vein through multiple short incisions. In 1905 Keller[50] advocated stripping the saphenous vein. For this he used a small wire, which stripped out the vein subcutaneously by completely everting it. A year later C. H. Mayo[72] introduced the vein-stripping operation that has been most widely used. Another modification of this operation is that devised by Babcock,[2] the principle in all cases being the complete extirpation of the great saphenous vein without extensive incision and dissection. Stripping the veins below the knee is impracticable or impossible because of the complicated and frequent branching of the veins.

The most satisfactory operative procedure finally evolved combined the stripping of the entire saphenous vein above the knee and the extirpation en masse by dissection of all the veins below the knee. This operation has been particularly advocated by Brooks,[11] Homans,[44-47] and many others. The complete operation is seldom used now, but it was the procedure of choice until a comparatively few years ago. It is interesting to note that Keller,[50, 51] who has been mentioned as one of the first to perfect an operation for vein stripping, described in 1924 an ingenious method of obliterating veins by suture. He used this on the varicosities of the lower leg in connection with the stripping of the saphenous vein. This procedure was essentially obliteration by thrombosis without extirpation. It had many advantages over the complete operation as described above, the chief objections to complete extirpation of the veins below the knee being prolonged hospitalization, the usual requirement of a general anesthetic, the danger of infection and embolism, and the danger of sloughing of skin flaps with delayed healing. The results of the complete operation (stripping of the saphenous vein and extirpation of the varicosities) were excellent. The magnitude of the procedure, however, was out of proportion to the seriousness of the condition.

HISTORY OF THE INJECTION TREATMENT OF VARICOSE VEINS

The treatment of varicosities by injecting into them some thrombus-producing agent is of course the same in principle as the ones previously described as methods used to obliterate the veins in situ. The actual in-

jection of varicose veins, however, depended upon and soon followed the invention of the Pravaz syringe in 1851. According to De Takats and Quint,[26] Pravaz injected perchloride of iron into varicose veins as early as 1853. In 1855 Desgranges[23] injected six cases of varicose veins with iodotannic solution. These injections were without accident, but apparently they were accompanied by a great deal of pain. In these early days of injection treatment there were probably a relatively small number of cases treated and these mostly in European clinics. Many different solutions were used, however, among them being alcohol in various solutions, Lugol's solution and phenol (De Takats, Quint, Tillotson,[24] and Crittenden[24] and Blackford[8]). These attempts fell into disrepute because of the severe reaction due to the toxicity and extremely irritating properties of these drugs. Sepsis may have played some part in these early attempts, but most of the solutions used at that time were strongly antiseptic.

In the latter 1870's there was developed in Continental clinics, particularly in Vienna, what Colt[17] refers to as "the era of perivenous injection." This applied not only to varicose veins, but to hemorrhoids, rectal prolapse, varicocele, etc. In these were used such solutions as ergot, alcohol, iron perchloride, chloral, phenol, etc. This type of treatment caused obliteration of veins in situ by fibrosis and was in reality much more similar to the older methods of obliteration previously mentioned than to the treatment we now speak of as injection treatment of veins. This type of treatment has of course been entirely given up in the case of varicose veins, but it is exactly the same as used today in the injection treatment of hemorrhoids and varicocele, except for the solutions used. It is rather important to emphasize that the obliteration of varicose veins by the intravenous injection of a thrombus-producing substance as practiced at the present time is entirely different in principle to the injection treatment of hemorrhoids, varicocele, hydrocele, and hernia. The latter is based upon exactly the same principle as treatment of varices by perivenous injection; viz., the setting up of a severe inflammatory process and the production of a large amount of scar tissue which by its contraction obliterates veins, venous sinuses, or serous cavities. The treatment of varicose veins by perivenous injection was found to be wholly unsatisfactory (Colt[17]).

The recent development of the treatment of varicose veins by injection was given impetus by Sicard,[90, 91] Model,[75] and Linser[60, 61] and their coworkers. Sicard and his coworkers and Linser discovered the sclerosing properties of drugs used in the treatment of syphilis. Sicard[91] in 1922 reported curing varices by the injection of sodium salicylate, later using other solutions. Linser[60] noted the excellent venous thrombosis and obliteration sometimes produced by the intravenous injection of bichloride of mercury in the treatment of syphilis. In 1925 he reported the successful treatment of varicose veins by this method and in

1925[61] reported the same thing, using 15 to 25 per cent sodium chloride. De Takats and Quint[26] give credit to these men for the development of the use of hypertonic solutions in the treatment of varicose veins. The therapeutic connection between varicose veins and syphilis was carried a step further by Borcherds,[9] who in 1927 reported curing a patient simultaneously of varicose veins and syphilis by intravenous injection of salvarsan. From this time on until recently, most of the advances in the injection treatment of varicose veins had to do almost entirely with the solutions used.

SOLUTIONS

Up to the time of Sicard and Linser the various solutions of mercuric salts had perhaps been the most widely used of all solutions, the chief objection to them being their toxicity which necessarily limited the dose given. There were other disadvantages, but these were largely shared by all solutions used until more recently. In 1919 Zirn[109] and others reported the successful use of 1 per cent sublimate of mercury, and in 1922 Fischer[38] reported the successful use of mercuric chloride. After the sclerosing effects of the various hypertonic solutions were reported in the early 20's and after the toxic effects of mercury had been emphasized, the solutions most commonly used were as follows:

1. The nontoxic hypertonic solutions of the various sugars and sodium chloride, the most popular of these becoming hypertonic sodium chloride, hypertonic glucose or dextrose, and mixtures of the hypertonic solutions of sodium chloride and glucose.

2. Sodium salicylate, sodium citrate, and solutions of some quinine salt, usually the hydrochloride, the latter being usually combined with urethane.

These solutions are put up by various pharmaceutical houses in various strengths and combinations and under various trade names.

The literature dealing with the use of the various solutions became enormous. In 1924 Troisier[101] reported using sodium citrate. In 1925 Bardy[3] recommended the use of quinine solution. Meisen[73] in 1927 recommended a mixture of sodium salicylate 25 per cent and sodium chloride 10 per cent. In the same year, McPheeters[66] favored 20 per cent sodium chloride. In 1928 Colt[17] reported the successful use of sodium salicylate with no bad results, except pain at the time of injection.

Alexander,[1] writing in 1926, reported good results with sodium salicylate. Schmier[89] in 1930 recommended sodium chloride, but warned against sloughing; in the same year Stuebner[94] reported excellent results using sodium salicylate. In the same year White[105] reported using quinine hydrochloride and urethane; Cattell[13] recommended quinine urethane solution in 1931, as did Lewis[59] in 1932, Kilbourne[53] in 1930, and Weeks and Mueller[104] in 1932.

In general the quinine urethane solution became more popular in European clinics than in American, and to a lesser extent this was true of sodium citrate and sodium salicylate. The hypertonic sugar and sodium chloride solutions and their combinations became more popular in America. These solutions were especially recommended by McPheeters[66-68] and De Takats[26-28] and many others up until fairly recently.

In general, quinine solutions have the advantage of causing a certain and very firm thrombus following the injection of a small amount of material. The disadvantages are their toxicity and the danger of sloughing when a small amount is injected outside the vein. The pain caused by their injection is also a disadvantage. This latter, however, is largely overcome by the addition of urethane to the solution. More or less the same advantages and disadvantages, each less marked, are found in the use of sodium citrate and sodium salicylate, the latter being more popular. Hypertonic sodium chloride is less efficient, apparently, as a thrombus producer than the above solutions, but is relatively nontoxic, although large doses have been followed by hematuria. Much larger amounts must be injected into the tissues to produce sloughing. It does not cause pain at the site of injection, but it does produce severe cramping pain in the extremity. Hypertonic sugar solutions are apparently still less efficient as thrombotic agents. They are nontoxic, however, may be injected into the tissues in relatively large amounts before producing a slough, and cause little or no pain at the time of injection. The higher concentrations are, however, likely to be somewhat harder to handle because of viscosity.

Various combinations of sugar and saline solutions seem to offset the disadvantages of each, but they still have the drawback of being rather inefficient as thrombus producers. Where these solutions have been used almost exclusively, occasional use of sodium citrate or quinine urethane solutions has been resorted to where the others failed to cause thrombosis (McPheeters,[69] De Takats[28]). Delater and Chailly[21] and Dunbar[32] also recommended using various solutions for different types of cases.

More recently the search for a more ideal sclerosing agent has centered mainly around the solutions of fatty acids or the salts of these acids. The most popular of these is now sodium morrhuate, usually the 5 per cent, occasionally the 10 per cent, solution. This solution was first referred to by Rogers and Murkegee[86] in 1919 in the treatment of leprosy, and in the same year Rogers[85] described its preparation. It was also used in the surgical treatment of tuberculosis. Its preparation was also described by Cutting[20] in 1926.

Higgins and Kittel[42] were the first to use sodium morrhuate in the injection treatment of varicose veins. It was further advocated as a sclerosing agent in the same year by Rogers.[87] In 1932 Tunick and Nach[102] reported 600 cases receiving injections of 5 per cent sodium morrhuate, using as a rule 2 to 5 c.c. They stated that its injection

caused no cramping pain, was the least likely of the solutions thus far used to cause sloughing, was nontoxic, and was a good thrombus producer.

In 1932 Kilbourne, Dodson, and Zeiler[56] published an important paper dealing with the toxicity, slough-producing properties, and bacterial action as related to phlebitis and embolism of various solutions used in the injection of varicose veins. They concluded that bichloride of mercury was too toxic; metaphane was less toxic and less likely to cause sloughing. Sodium chloride and glucose were nontoxic, relatively painless, and less likely to cause sloughing than the quinine and salicylate solutions, but they had the disadvantage of being nonbactericidal. They found sodium salicylate to be an excellent thrombus producer, but it produced cramping pain at the time of injection and was likely to produce sloughs. Lithium salicylate was painless but more toxic. They found quinine urethane excellent except for its toxicity and the danger of sloughing. They found sodium morrhuate to be nontoxic, less likely to produce serious sloughing, efficient as a thrombus producer, but having the disadvantage of being nonbactericidal.

Smith[92, 93] endorsed sodium morrhuate and reported more than 2,000 injections with no sloughs or other complications and a lower incidence of recurrence. Tournay[99] in 1933 favored the use of sodium morrhuate. However, although sodium morrhuate produced an excellent thrombus, was nontoxic, painless, and less likely to produce a slough, it was found to have one objection—some individuals were sensitive to it. Warning of the danger of this was given by Zimmerman.[108]

In 1934 and 1935 Ochsner and Mahorner[70, 78] published the results of experimental work on twenty-nine solutions. They found that sodium morrhuate injected into the veins of dogs was the most efficient thrombus-producing solution of the twenty-nine solutions tried. Sodium gynocardate was second. As a whole, they found sodium morrhuate Searle to be the best solution tried. In 1934 Cooper[19] published a report of more than 35,000 injections of various solutions in 3,164 cases. He reported 293 cases of extensive and recurrent varicose veins treated by preliminary ligation and subsequent injections. Of the various solutions he had tried, he recommended sodium morrhuate.

In 1935 Colt and others[18] preferred salicylate solutions and found sodium morrhuate less effective. In the same year Christie[16] advocated the use of quinine urethane, sodium salicylate, and 5 per cent sodium morrhuate. He issued a warning against the danger of the latter, which he stated had caused fatalities. Specific fatal cases were not, however, reported. In the same year Barker[4] stated that sodium morrhuate was preferred at the Mayo Clinic. Praver and Becker[82] in 1935 studied the sensitization phenomena following the injection of sodium morrhuate. They reported that 7 of 175 patients receiving 783 injections of sodium morrhuate showed sensitization reactions. Probstein[83]

in 1936 also reported several cases of sensitization reactions but no deaths following the use of sodium morrhuate. In the same year Willauer[106] also recommended sodium morrhuate.

Froehlich and Henrikson[39] in 1935 recommended sodium ricinoleate, as did Postlethwaite[80] the following year. They used a 2 per cent solution and found it to be effective at thrombus production and less likely to produce reactions than sodium morrhuate.

Recently Biegeleisen[7] has published the results of evaluating four new fatty acid solutions. He concludes: "The use of fatty acid salt solutions is an advance toward safer and more efficient sclerosing therapy." He presents four new solutions: (1) moru-quin, a loose chemical composition of sodium morrhuate and quinine, which he believes is a new and useful solution; (2) oleate quinine, combining potassium oleate and quinine, a stable mixture which he finds to have a uniform sclerosing power; (3) sylnasol, the fatty acid salt of psyllium seed oil, which he finds resembles sodium morrhuate in its action on veins; and (4) monoethanolamine oleate, a synthetic compound which he believes at present to be the most efficient sclerosing agent for the treatment of varicose veins.

Very recently Johnston,[49] in a paper dealing with ligation and retrograde injection, states that he uses 5 per cent sodium ricinoleate in doses of 4 to 10 c.c.

The situation in regard to solutions can be summed up as follows: As a whole the solutions of the salts of fatty acids seem to have more advantages and fewer disadvantages than any of the previous older solutions used. Of these, sodium morrhuate has been most widely used and has been found on the whole to be very satisfactory. It is perhaps more popular in America than abroad (Riddoch[84]). It has been shown both experimentally and clinically to be superior to other solutions, with the possible exception of sodium ricinoleate. The latter, however, has been less widely used. The newer solutions described by Biegeleisen[7] are extremely interesting and we hope may prove satisfactory. Certainly sodium morrhuate is not an ideal solution, but it seems to be the best that has been used so extensively. Its greatest disadvantage lies in the sensitization reactions occasionally seen. In our experience these have been extremely rare and not at all alarming. We do not believe that the disadvantage pointed out by Kilbourne and his coworkers,[56] namely, its not being bactericidal, should counteract other obvious advantages. Proper technique should make the danger of contamination as little as in any intravenous therapy. Proper selection of cases will largely rule out injecting veins already the site of a thrombophlebitis.

MOST ACCEPTABLE METHODS OF TREATMENT AT THE PRESENT TIME

The recent advances in the treatment of varicose veins of most importance have to do with various combinations of operative and injec-

tion therapy. In many therapeutic procedures no attempt has been made to correct the fundamental conditions in varicose veins. This is, in large measure, the reason for failures with the older operative procedure and the injection treatment.

The superficial veins of the lower extremity dilate because of increased pressure. This may be due to obstruction of the deep venous system, such as occurs in thrombophlebitis. In this case the dilatation is merely compensatory. This type, of course, is rarely amenable to treatment. The same thing may occur during pregnancy because of pelvic pressure obstructing the venous return from the legs. In this case the pressure, acting on veins with relatively weak walls, gradually breaks down all elasticity of the vein walls and the dilatation becomes permanent. When this occurs, the valves also become incompetent. In other cases there is no obstruction, but long periods on the feet with resulting increase in venous pressure in the lower extremities cause dilatation of the superficial venous system. An especially important factor is the inherent weakness of the veins in some individuals. Once this dilatation has gone far enough to cause the valves in the great saphenous vein to become permanently incompetent, the condition is self-propagating.

It has been pointed out by many observers that what actually occurs in the classical case of varicosities of the great saphenous system with incompetent saphenous valves is not an increase in pressure or stasis but an actual reversal in the direction of the flow.

Brodie[10] in 1846 described the reflux of reverse flow in the saphenous vein which occurs after emptying the vein, obstructing it, and quickly removing the obstruction. This was described by Trendelenburg some fifty-five years later and the sign given his name. Brooks[11] emphasized the evident actual reverse flow down the saphenous and into the deep circulation. In 1927 Berntsen[5] pointed out that the surgical treatment, to be effective, must keep these fundamental principles in mind. The pathology of abnormality of the venous circulation in the varicose extremity was beautifully described by De Takats, Quint, and coworkers[24] in 1929. Schmier[89] in 1930 demonstrated the reverse flow in the venous circulation by intravenous injection of substances opaque to x-rays and following their progress radiographically. This subject has been reviewed comprehensively by McPheeters.[69]

It would seem fairly obvious that if a saphenous vein were dilated and its valves incompetent, and one injected or excised the varicosities below the knee, regardless of how well this were done, one would get subsequent dilatation of other veins emptying into the saphenous. One cannot remove, nor is it desirable to remove, all the superficial venous system of the leg. As long as there are any veins, no matter how small, which communicate with a larger vein having increased venous pressure, they will dilate and become varicosities.

This does not apply only to varicosities of the great saphenous system, but these are the most frequent and most typical. The same principle applies to varicosities of the small saphenous vein. This vein occasionally empties into the great saphenous and the two systems become one. More frequently it empties into the deep circulation near the popliteal space. If the valves in this communication are incompetent, the same reverse flow occurs. In any group of varicosities communicating with the deeper circulation through a dilated channel with incompetent valves, the same principle holds.

It is well known and has been shown histologically that after the formation of even the firmest thrombus by injection there is some retraction of the thrombus and recanalization. Obviously, if these small veins within veins are connected with a large vein in which there is an increased pressure of reverse flow, they will eventually dilate and the varicosities will recur.

Lufkin and McPheeters[62, 69] and others pointed out this retraction and recanalization. McAusland[64] points out that no treatment cures the tendency toward the development of varicose veins. Hence, failures are not necessarily recurrences of persisting varices, but they may be the formation of new ones.

This recanalization does not depend upon the solutions used. Quinine and urethane solution has always been considered an efficient thrombus producer, yet Faxon[35] reported 63 per cent of recurrences after the use of this solution; Howard[48] reports 100 per cent recanalization. It seems probable that the rôle of recanalization in recurrence of varicosities has been overemphasized and the formation of varicosities from previously uninvolved veins not sufficiently emphasized. Even when using the now more or less standard technique of injection, one of the better solutions and properly selected cases, the injection treatment when carried out alone often yields very disappointing results from the standpoint of recurrence. In an attempt to overcome this deficiency, various types of operations combined with injection have been used during the past several years.

For a period of between two and three years, beginning in 1930, the favored procedure in the treatment of varicose veins in the Vanderbilt University Hospital was the complete removal of the saphenous vein, followed by injection of varicosities below the knee. This procedure was carried out in all cases where incompetence of the saphenous valves with backflow in this vein was demonstrated. The vein was divided at its junction with the femoral after dissecting out and dividing the higher branches. It was then stripped out with a Mayo vein stripper as far as possible, usually to a point slightly below the knee. Varicosities of the leg were later obliterated by injection. The number of cases treated in this manner was relatively small, but the results were highly satisfac-

tory. This type of treatment has been for the most part discarded in favor of the simpler procedure of ligation of the saphenous followed by injection of the varicosities.

Ligation of the saphenous vein in conjunction with injection was first carried out and reported by Tavel[96] in 1904. He ligated the saphenous vein and injected 5 per cent carbolic acid below the ligation. Thus not only was he the first to combine ligation and injection, but he was also the first to carry out what we now know as retrograde injection, which will be considered later.

To De Takats[27] goes the credit for emphasizing the importance of high ligation of the saphenous vein and its popularization. He first advocated this as an ambulatory procedure to be used in conjunction with injection of varicose veins in 1930. He stated that it would definitely decrease the danger of embolus after injection and obliteration of the saphenous vein. He also repeatedly pointed out its usefulness in breaking dependably and permanently the vicious cycle of reverse circulation and removing the head of pressure from the saphenous tributaries. He[27] recorded that 22 to 72 per cent of recurrences followed the Trendelenburg operation, but he believed the results would be excellent if combined with obliteration by injection. However, the reason that the Trendelenburg operation was not more satisfactory was probably due to the fact that it was not sufficiently high, the ligation being carried out usually below the first branches of the saphenous vein. The importance of the high interruption of the saphenous vein has been pointed out repeatedly by Homans[44-47] in connection with operative treatment of varicose veins. It is probably even more important when simple ligation or division between ligatures is carried out in conjunction with injection. This has been pointed out by De Takats and his coworkers,[27, 28, 30] Hawkes and Borsher,[41] Howard,[48] Edwards,[33] Willauer,[106] and others.

In cases of marked varicosities of the saphenous system, the saphenous vein itself may not be the only incompetent communication with the deeper circulation. The communicating vein of the small saphenous may be at fault. Occasionally there is a large communicating vein with incompetent valves at about the junction of the middle and lower thirds of the saphenous vein.

In general, if the valves of the great saphenous vein are not competent, only injection is carried out. This is probably the cause for many recurrences. De Takats[27] in 1930 stated that he advocated operating radically on 4 per cent of cases with incompetent communicating veins. Other men, among them Willauer,[106] have stressed the advisability of ligating the small saphenous vein at its junction with the popliteal when this system is involved. Other communicating veins, such as the one mentioned above, should be ligated when at fault. The detection of these communications which should be obstructed has been emphasized and made easier by the multiple tourniquet test devised by Mahorner and Ochsner.[71]

It would seem that ligation has the advantage over stripping out of the saphenous vein, being a much more minor procedure. Removal of the saphenous vein, requiring longer hospitalization, is usually performed under general anesthesia and opens up a wide field to bacterial invasion. Undoubtedly the danger of anastomosis around a ligature and recurrence is greater after ligation than after extirpation of the entire saphenous vein. It is, however, doubtful if it is justified in most cases. Until recently, however, we felt that it should be carried out in a few particularly recurrent cases.

Perhaps the most valuable recent contribution to the therapy of varicose veins is the introduction of so-called retrograde injection. It seems at the present to combine the advantages of extirpation of the saphenous vein and simple ligation and not to have many of the disadvantages of either procedure. As previously mentioned, Tavel[96] was the first to carry out retrograde injection. It was more recently introduced, in 1927, by Moszkowicz.[76] To Faxon[36] should go the credit for introducing it into this country and aptly emphasizing its advantages. It has also been recommended by Fantus,[34] Swinton,[95] Haggard and Kirtley,[40] and very recently Johnston.[49] The procedure consists simply of division between ligatures of the saphenous vein at its junction with the femoral. Into the distal stump a sclerosing solution is then injected. As pointed out by Johnston, the obliteration of the entire saphenous vein, which is accomplished by this method, should result in fewer recurrences than simple ligation of the vein followed by injection from below. The mortality or morbidity should not be increased over that of simple ligation.

At the present time the most satisfactory procedures to be carried out in the treatment of varicose veins are as follows: Simple injection should be carried out in isolated varicosities without demonstrable incompetent communications with the deep circulation. In cases of incompetent saphenous valves, the saphenous vein should be divided between ligatures at its junction with the femoral, after dissecting out and dividing between ligatures all of the upper branches. This is to be followed by retrograde injection. In all cases, careful search for other incompetent communicating veins should be made. If these are found, they should be divided between ligatures at their source and retrograde injection carried out from this point.

Somewhat special groups of patients with varicose veins may call for some modification of this treatment.

One of the most frequent types of patient to seek relief from varicose veins is the pregnant woman. Pregnancy is one of the most common causes of the development of permanent varicosities, and frequently varicosities are the chief source of suffering during pregnancy. The knowledge that varicosities can be treated by injection during pregnancy is not new. McPheeters[69] has long advocated such treatment,

as have De Takats[26, 27] and others. Treatment of varicosities in this type of case, however, apparently needs emphasis.

As late as 1935, Cheatham and Peck[15] stated that the idea is still not prevalent that such varicosities should be treated. They pointed out that the textbooks of both DeLee and Williams state that little can be done for varicosities during pregnancy.

For several years we have made it a practice to treat varicose veins during pregnancy, up to about the seventh month, much as we treat varicose veins in any other case. If ligation is indicated, this is carried out. After ligation or if it is not necessary, the larger and more troublesome veins are obliterated by injection. More recently we have employed retrograde injection in these as in other patients. No complications have occurred to date. We are, however, less assiduous in attempting to obliterate all dilated veins. Except when treated very early in pregnancy, only the ones causing symptoms are obliterated. The patient returns six to eight weeks after delivery for further treatment. Usually, if ligation has been done, the smaller varicosities, and many times the larger ones, will entirely disappear after delivery.

A number of patients have been observed during pregnancy who had previously had high saphenous ligation either during or between former pregnancies. These patients, of course, have the same amount of venous obstruction acting as a causative agent as previously. They do not, however, develop varicosities of the saphenous system. They frequently do develop varicosities of the upper thigh, particularly of the posterior surface; these veins anastomose with veins above the saphenous and femoral junction, very frequently the gluteal veins. If these varicosities cause symptoms, they should be injected. Labial varicosities can be injected. It is, however, a somewhat more difficult procedure and in our experience only rarely necessary or desirable.

Another special group of cases is that group in which varicosities occur in the very old or infirm. Usually only the injection treatment is carried out in these patients, and this is confined to the larger varicosities. If they recur, they may be re-injected.

TECHNIQUE

So far we have not discussed the technique of any therapeutic procedure in detail. This has been covered very well indeed by many authors. In those cases where it may still seem advantageous to extirpate the entire saphenous vein, it is best removed by stripping operation, using the Mayo vein stripper. The most important details include extraordinary care in regard to asepsis and hemostasis. One should be certain of dividing the vein at its junction with the femoral and should dissect out and divide all of the upper branches.

Formerly it was believed that the sclerosing solution should ideally be injected into a vein as nearly empty as possible, and immediately

after injection the vein should be maintained in this collapsed state by a supporting bandage or dressing. This it was hoped would cause the vein walls to unite more or less directly by fibrosis with the maximum obliteration and minimum thrombus formation. It is also true that a better obliteration results if the solution reaches the vein in high concentration, thus assuring more damage to the intima. With this in view, various methods of occluding portions of a vein from the surrounding ones were devised. In the case of the long varicosities, a segment between two tourniquets was injected, only one injection being made if one of the toxic solutions were used. In the case of the hypertonic sugar and salt solutions, multiple injections were made between many tourniquets applied simultaneously, as described by McPheeters.[69] For more isolated superficial veins, various types of occluders were devised to keep the solutions localized longer (McPheeters,[69] Potts,[81] Theis[98]). Because a sclerosing solution reaches the intima in a higher concentration if the vein is empty, injection in the horizontal position has been most popular. For this reason various methods have been devised to enable the needle to enter a full vein and to enable the injection to be made into an empty one. Occluders, tourniquets, and special tables, such as the one devised by Stuebner,[94] were used for this purpose.

It is no longer held that veins should be kept collapsed after injection. In fact a better obliteration follows a full thrombus (McPheeters[69]). The wearing of some light supporting bandage after injection, however, makes the patient distinctly more comfortable.

The value of limiting the sclerosing solution to a given area was especially useful when using the glucose saline solutions. The sclerosing power of these solutions was their weakest point. This is not necessary with sodium morrhuate. We still prefer to inject varicose veins with the patient lying down; in many clinics they are injected with the patient standing. We use no tourniquets, constrictors, etc., preferring that the solution go as far as possible and the maximum extent of the thrombosis be obtained. The special equipment needed includes Luer lock syringes or syringes of some similar type, usually 5 and 10 c.c., and several sizes of needles with short bevels. It is a mistake to use too small a needle, as this slows up injection and causes unnecessary dilution of the solution.

Very small cutaneous veins, so-called spiders or sunbursts, can be effectively obliterated by using a very small needle and by injecting a very small amount of fluid, preferably 5 per cent sodium morrhuate (Biegeleisen[6]). The very small ones may be obliterated with somewhat less danger of slough by injecting small amounts of sodium morrhuate mixed with air, i.e., bubbles or even small amounts of air.

The technique of saphenous ligation has been especially described by De Takats[27, 30] and Faxon,[36] the most important points being the use

of local anesthesia, strict attention to asepsis and hemostasis, and careful dissection to insure ligation above the three upper branches: the superficial iliac circumflex, the superficial epigastric, and the external pudendal. Recently Johnston[49] has pointed out the advisability of carefully dissecting out and dividing these branches. Their position is variable and unless this is done they may be missed. This is particularly true in the occasional cases where they actually empty directly into the femoral vein.

We agree with Faxon[36] that the operation is not trivial and prefer always to hospitalize a patient for twenty-four to forty-eight hours for this operation. This is largely due to fear of infection. The incision is made in a region requiring special cleansing of the skin. An infection at the fossa ovalis is indeed a serious thing. An added risk is due to the fact that so many of these patients have had varicose ulcers with residual infection in the lymph nodes which lie in the operative field.

The technique of division of the saphenous vein and retrograde injection has been very adequately described by Faxon[36] and very recently by Johnston.[49] The vein is divided between ligatures as in simple ligation. The sclerosing solution is then injected into the distal stump. We have seen no necessity for using cannulas as described by the above authors. An ordinary syringe with a fairly large needle suffices. Both ends are ligated with suture or transfixion ligatures. If any of the sclerosing solution has been spilled, the wound should be gently flushed out with salt solution. The wound is closed without drainage with interrupted silk sutures. Johnston has pointed out the danger of contamination of these wounds. For this reason we use a very fine cuticular skin suture of fine silk. The wounds are then covered with silver foil as an added protection and a very tight outer dressing applied. If 5 per cent sodium morrhuate is used, as much as 10 c.c. may be injected into a saphenous vein without undue reaction. In cases of extensive thrombosis with considerable local reaction, the patients are made more comfortable by wearing light elastic bandages for a few days over the thrombosed veins. The patients are encouraged, but not forced, to walk.

CONTRAINDICATIONS TO TREATMENT

All operative procedures may be contraindicated because of advanced debilitation or senility. Extreme disability should also be a contraindication to extensive injection treatment. It is sometimes justifiable, however, to obliterate those veins directly causing symptoms. This is particularly true in the case of ulcers. These may often be healed, at least for some time, by simply injecting the vein or veins in the vicinity of the ulcer.

The chief contraindication to any type of therapy is, of course, thrombophlebitis. If the deep circulation has been the site of extensive thrombophlebitis in the past, it is unlikely that the patient will be bene-

fited by any therapy, and great harm may be done. In addition, if injection is carried out, the inflammatory process may be activated, the process spread, and embolism may occur (De Takats[29]). The latter is true of phlebitis confined to the superficial system as well as to thrombophlebitis of the deep veins. Varicosities often become the site of a spontaneous thrombophlebitis. Zimmerman[108] mentions a case of thromboangiitis obliterans activated by injection. Certainly no patient with impaired arterial circulation in the extremities should have varicosities obliterated.

One of the greatest drawbacks to the injection treatment of varicose veins is the ease with which it is carried out. It is used by many without sufficient understanding of the disease, proper examination of the patient, or proper selection of cases. Although the technique of injection is simple, accidents may occur in inexperienced hands. Large sloughs from fluids injected outside of the vein are the most common complications. We have observed one patient with gangrene of the distal portion of the foot following accidental injection of the anterior tibial artery.

RESULTS OBTAINED

It may be stated briefly that the earlier operative procedures were relatively unsuccessful. Later the complete extirpation of the saphenous vein and the varicosities below the knee gave excellent results. This operation, however, was of too great a magnitude for the disease. This was especially true from an economic standpoint, the treatment requiring relatively long hospitalization following a major operation.

The early reports of the injection treatment were almost universally favorable. Subsequently when the more irritating and toxic solutions were used, there were many reports of complications, such as sloughs and pain; even an occasional death was reported. With more innocuous solutions coming into use again, the only complication reported was sloughing.

Cattell[12] also discussed and reported such complications as burning after injection, nausea, and sloughing. Schmier[89] warns against the danger of slough. Kilbourne[54-56] also discusses the treatment of post-injection sloughs and other complications; he emphasizes the importance of reporting complications in order that the real dangers of this form of treatment may be understood.

In 1931 De Takats[28] pointed out the danger of toxic solutions, such as mercury, the salicylates, and quinine. The following year[29] he warned against injecting veins that are at the time or have been in the past the site of thrombophlebitis. Colt,[17] in his article dealing with the history of the evolution of injection treatment of varicose veins, also emphasizes the complications following the employment of the more toxic and irritating solutions.

As previously stated, the immediate results of the injection treatment seemed excellent; that is, the veins were quickly and efficiently thrombosed and obliterated and the patients were relieved of their symptoms. With the development of better technique and more satisfactory solutions, the danger of toxic reactions and the frequency of sloughs also diminished. The danger of embolism, however, was still feared.

At the first glance it would seem that the production of a massive venous thrombosis would result in an occasional embolic death. This is the case, of course, in the ordinary septic thrombophlebitis. In the case of injection obliteration of varicosities, however, the facts have been proved otherwise. Statistics show that there is very little danger of embolism following the injection treatment of varicose veins. Histologic studies on veins following injection show the reason for this.

In spite of the fact that there was a gradual increase in the number of cases of injection recorded, the fatalities were few, so that it would seem apparent that the danger was slight; and when actual comparisons were made, it was found to be less than following operative treatment. Bardy[3] in 1925 stated that operative therapy was satisfactory but that the danger from embolus was greater than with injection. In the same year Dunbar[32] stated that there was more danger of embolism following operation than following injection, although in both instances the results were satisfactory.

In 1927 Meisen[73] reported 500 cases treated with 2,224 injections without any deaths. In the same year McPheeters[66] reported 31 cases with 180 injections without complications and stated that the mortality was lower than with operation. In the following year McPheeters[67] collected a series of 53,000 injections with 7 deaths, a mortality of 0.033 per cent. In the following year De Takats[25] reported 1,000 injections without a death and emphasized the low mortality of the method. Kilbourne,[53] in 1929, also reported that in a series of 4,607 cases operated upon there was 1 death from embolism in every 250 cases. In 1930 Pennoyer[79] found 4 authentic cases of death from pulmonary embolism in 400,000 reported injections. In 1932 Lewis[59] reported 848 cases receiving 3,532 injections with no deaths. In 1934 Kilbourne[57] reported 20,000 injections with no mortality. He studied 20 reported deaths after injection and concluded that the majority were due to bacteremia caused by contamination at the time of injection or the lighting up of an old thrombophlebitis. These factors can be eliminated by proper technique and proper selection of cases. De Takats[29] also believes that injection of veins in patients who have previously had thrombophlebitis is the most important factor in the formation of emboli and death.

It is also interesting that Kilbourne[57] points out in this connection that there is less danger of infection developing after the use of solutions which are definitely bactericidal. Most of the solutions now in use are

only weakly or not at all bactericidal. This should not be of importance, since injections should not be carried out if dependence on asepsis must rest with the bactericidal power of the solutions used. In 1935 Theis[98] reported 14,000 injections in 882 patients with 1 fatal pulmonary embolism.

The reasons for the relative safety of the injection method are explained by histologic and pathologic findings. When any of the common operative procedures for treating varicose veins are carried out, blood clots are left in close proximity to open veins and ligated veins form thrombi in their stumps. These are fairly firm clots but they are not dependent on injury to intima for their formation. In postoperative thrombophlebitis, simple stagnation of blood is probably the most important factor. Hence the thrombus is not densely adherent to the intima and therefore at times is loosened into the blood stream. In the case of bacterial thrombophlebitis this danger is much greater. The thrombus is formed by the action of the bacteria on the stagnant blood stream and upon the intima. After the formation of this thrombus, however, the bacterial action is to break down and liquefy it, thus replacing the original and rather firm clot with a mass of small thrombi, loosely adherent in a semifluid state. This furnishes the ideal basis for embolism.

In the case of thrombosis of a vein following the injection of some highly irritating substance, the thrombus forms as a result of severe injury to the intima. The intimal cells first swell and often rupture; the thrombosis is laid down upon this injured area and becomes densely adherent to it. Instead of subsequent liquefaction, the thrombus quickly undergoes organization, the fibroblasts growing directly from the vein wall into the clot. Thus there is less danger of emboli breaking off and entering the blood stream.

In 1894 Delore,[22] in defending the treatment of varicose veins by injection of iodotannic solution, pointed out that the resulting condition was a chemical endophlebitis and that the thrombus depended on this. Since that time, histologic studies, both on the veins of experimental animals after injection and on the veins of patients excised after injection, have been carried out by various workers. All show the same type of thrombus following injury to the intima, resulting in fibrosis of the clot and vein wall. The following have contributed to this important phase in understanding the injection treatment of varicose veins: Nicholson,[77] Lehman,[58] De Takats,[25, 27] Lufkin and McPheeters,[62] McPheeters,[69] and Mahorner and Ochsner.[70, 78]

With a fairly definite understanding as to the comparatively slight risks involved in the injection treatment, the next most important consideration naturally was and is that of results from the standpoint of cure and relief of symptoms. As stated previously, most of the earlier reports were optimistic. In most hands the injection of varicose veins,

especially using the earlier and more irritating solutions, resulted practically always in thrombosis and rapid obliteration of the veins. After a large number of patients had been treated and followed in some clinics over a period of time, however, reports as to the results became very variable, ranging from the usual optimistic to those frankly pessimistic. In Meisen's[73] 500 cases there were 14 recurrent cases which had been operated upon and 10 recurrent cases in an additional series of 50 cases; thus 24 cases were recurrences following previous operation. He believes the rate of recurrence to be lower after the injection treatment. De Takats[28] in 1931 reported 10 per cent recurrences. Kettel[52] in 1932 reported 400 cases with 94.5 per cent immediate cures and 91.5 per cent cures after one year. One-third of the latter group showed some recurrences without symptoms. In 1932 Lewis[59] stated that recurrences were infrequent and pointed out that recurrences and the formation of new varicosities should not be confused. In the same year Wymer[107] emphasized the relative infrequency of recurrences after injection treatment. In 1933 De Tarnowsky[31] reported 63 per cent cured. In the same year Faxon[35] at the other extreme reported 63 per cent recurrences in 1.4 years. The following year McKinstry,[65] in reviewing the work for the previous year, 1933, commented upon the marked variations in the reports as far as results were concerned and the inadequacy of our knowledge of the subject.

All of the above results apply to cases treated with injection alone. It is believed at the present time that these results are being greatly improved upon by combining obstruction of communications with the deep circulation with obliteration by injection.

As stated previously, our own results combining extirpation of the saphenous vein with injection were excellent. Recently we have tried to trace all of the patients treated in this manner. Most of them were treated between four and five years ago. Many of them have not been traced to date. There have been no recurrences in the ones examined which were marked enough to cause symptoms or to demand re-injection.

In 1933 De Takats and Quillin[30] reported 170 recurrences in patients treated with combined ligation and injection. Hawkes and Borsher[41] stated that injection combined with ligation in the lower or midportion of the thigh had been followed by recurrence, but they had no recurrences in 10 cases where high or fossa ovalis ligation was carried out. Howard[48] in 1934 reported his results one year and twenty-nine months after treatment by combined ligation and injection. He found there was some recanalization, but that 77 per cent were cosmetically cured; he believed the best results were obtained by this treatment. In the same year Edwards[33] reported excellent results and fewer injections necessary after high ligation of the saphenous vein. In 1935 Ferguson[37] advocated combined ligation and injection, reporting good results and no complications.

Retrograde injection has not been in use a sufficient length of time for the later results to be known. The immediate results in our own cases have been excellent. We have no complications with this method. All the patients have had their period of treatment shortened because of the fewer number of subsequent injections required. Some of these patients have not required any further injections, all varices being obliterated by the retrograde injection. Faxon,[36] Haggard and Kirtley,[40] and Johnston[49] have also reported good immediate results.

SUMMARY AND CONCLUSIONS

In summary, it may be said that the modern or present-day treatment of varicose veins consists primarily of occluding them by the injection of sclerosing solutions. The combination of this method with ligation of the saphenous or incompetent communicating veins, where indicated, is the treatment of choice. We believe that the method of retrograde injection is of great help in carrying out this treatment. At present, 5 per cent sodium morrhuate seems to be the solution of choice. Other solutions of fatty acid salts or these salts combined with some other compound may prove to be superior.

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Review of Recent Meetings

REVIEW OF THE THIRTY-FOURTH ANNUAL MEETING OF THE AMERICAN UROLOGICAL ASSOCIATION, MINNEAPOLIS, MINN., JUNE 28-JULY 1, 1937

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ATTENDANCE at the meeting was unusually large considering its location in the Middle-West (856 registering). A feature of the meeting was the largest scientific exhibit in the history of the Association, more than sixty exhibits being presented.

The first half day was devoted to a symposium on hydronephrosis. The common extrinsic causes thereof were enumerated by **Dr. Ray M. Bobbitt,** of Seattle, who feels that accessory vessels to the lower pole of the kidney cause obstruction only if accompanied by periureteral fibrosis or ptosis. **Dr. C. P. Mathe,** of San Francisco, discussed intrinsic causes, grouping them as congenital, acquired, and traumatic.

Dr. E. G. Crabtree, of Boston, summarized current knowledge of hydronephrosis of pregnancy. He stated that the underlying cause is compression of the ureters by the enlarging uterus, but that this effect is intensified by some endocrine factor which diminishes the tone of the renal pelves and ureters; and he reviewed the experimental evidence supporting this contention. Regression ordinarily begins three weeks after delivery and is complete in seven weeks.

Dr. Roy B. Henline and **Dr. James L. Bray,** of New York, advocated the use of serial retrograde pyelograms to determine the emptying time of the renal pelvis in the presence of questionable hydronephrosis; a kidney requiring more than ten minutes to expel the contrast medium is regarded as abnormal.

The next four papers reflected the increasing optimism with which operations for the relief of obstruction at the ureteropelvic junction are now regarded. Although the first such operation was performed by Trendelenburg in 1886, most surgeons are still inclined to remove kidneys which have become hydronephrotic as the result of obstruction from accessory renal vessels, periureteral fibrosis, and stricture.

Dr. Samuel Lubash, of New York, described a modification of Kuster's division and reimplantation of the ureter which has worked well in the experimental animal and in one human case.

Dr. Frederick E. B. Foley, of St. Paul, reported his experience in 20 operations in 19 cases with his Y-plasty, in which he takes a triangular flap from the inferior surface of the renal pelvis, draws it into a vertical incision in the lateral wall of the ureter, and pulls the junction into a dependent position. Accurate suture is most important. He reviewed 20 operations on 19 patients with almost uniformly good results; all had been followed for considerable periods by means of retrograde pyelograms. This procedure is theoretically superior to previous methods in that it neither interrupts the continuity of the pelvis and ureter nor causes buckling at the site of repair.

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Dr. Waltman Walters, Dr. Hugh Cabot, and **Dr. James T. Priestley,** of Rochester, Minn., reported on 46 plastic operations, chiefly lateral pelvioureterostomy, and division and reimplantation of the ureter, for the same condition; 15 of these subsequently required nephrectomy; 33 patients were cured or improved. Most of the secondary nephrectomies were done early in their experience; they attribute the recent improvement in results to the use of temporary nephrostomy following the plastic procedure.

Dr. William C. Quinby, of Boston, and **Dr. James C. Sargent,** of Milwaukee, both obtained favorable results with section and reimplantation of the ureter (Kuster's operation), the former having several patients well more than ten years after operation.

Dr. Nelse F. Ockerblad and **Dr. Hjalmar E. Carlson,** of Kansas City, described the distribution of ureteral pain as determined by stimulation of the ureter through a catheter electrode. They believe that the typical location of ureteral pain is a great aid in distinguishing lesions of the ureter from intraperitoneal disease.

Dr. J. Duane Miller and **Dr. William J. Butler** of Grand Rapids, Mich., stated that cystograms and urethragrams are of great value in determining the cause of urinary incontinence in the female, and that they serve as valuable guides to the type of repair required.

Dr. George M. Fister, of Ogden, Utah, drew an analogy between interstitial cystitis (Hunner ulcer) and lupus erythematosus, and reported a case in which great benefit had followed the parenteral injection of preparations of bismuth and of gold sodium thiosulphate. Any useful addition to the therapy of this most troublesome condition will be warmly welcomed.

Dr. C. D. Creevy, of Minneapolis, concluded that the factor which makes emptying of the chronically distended bladder potentially dangerous is not, as is commonly claimed, the rate at which the urine is withdrawn, but rather the introduction of infection by the catheter; he supported this contention by autopsy records, showing that patients whose bladders were emptied suddenly died of infection, and that the rate of emptying the bladder did not influence either the pathologic picture in fatal cases or the mortality in all cases. Gradual emptying of the bladder should be replaced by aseptic catheterization.

The second morning was given over to a symposium on renal tumors starting with an historical review by **Dr. Judson Bennett Gilbert,** of Schenectady.

Dr. E. T. Bell, of Minneapolis, presented a sample and logical classification of renal tumors.

Calcification in renal neoplasms was described by **Dr. George F. Cahill,** of New York, in 15 per cent of 84 renal neoplasms. It is usually secondary to hemorrhage or necrosis and is often associated with or followed by dissemination of the tumor.

Dr. Walter H. McNeill, Jr., of New York, reported a case of Wilms' tumor of the kidney treated by x-ray alone (10,000 R. over a period of more than a year), with apparent recovery at the present time. He cited the case of Coley and Ritchey who was well three years after irradiation alone, but concluded that the best prognosis is afforded by irradiation followed by nephrectomy.

Dr. Archie L. Dean, of New York, said that renal tumors in the adult are radioresistant as far as the possibility of cure is concerned, but that many are radiosensitive in that they may diminish materially in size after irradiation. It is to be remembered that irradiation injures the walls of the blood vessels in the tumor and may lead to infarction and severe bleeding. Preoperative irradiation

may reduce the size of a tumor but will not make operable a tumor which is inoperable because of local extension or metastasis. Technique was discussed.

Dr. George Gilbert Smith, of Boston, discussed the surgery of renal neoplasms. He employs a lumbar incision for small tumors and for those of uncertain nature. The transperitoneal approach is used for large growths because it permits earlier control of the blood supply and thus lessens hemorrhage at operation.

The second afternoon was occupied by the presidential address and the Guiteras lecture. **Dr. Gilbert Thomas,** of Minneapolis, the president, reported the results of a study of a large series of cases of renal tuberculosis; he took the position that renal tuberculosis is a slowly progressing disorder in the treatment of which conservatism is essential. It is advisable to treat the tuberculosis with general hygienic measures, meanwhile taking as long a time as is necessary to determine the condition of the other kidney; when it has been shown to be free of disease, the tuberculous kidney should be removed.

Occasionally, under hygienic measures alone, a minimal renal tuberculosis appears to heal with the disappearance of bacilli from the urine; bilateral disease may so improve as to permit the patient to return to work.

In the man, however, unilateral destructive lesions require surgery after deliberate rather than hasty study.

Dr. W. F. Braasch, of Rochester, Minn., in the Guiteras lecture made an exhaustive review of pyelonephritis. He emphasized the frequency of the colon bacillus as the primary invader and the importance of stasis as a predisposing factor, and pointed out that infection may lead to stasis by producing toxic atony, and later fibrosis of the renal pelvis and ureter.

He discussed the relationship of infection, especially with staphylococci, to stone formation. Retrograde pyelography is of great value in identifying the cicatricial changes of pyelonephritis and the destructive lesions of tuberculosis.

Modern conceptions of chemotherapy were described, with special emphasis upon the value of the ketogenic diet and of mandelic acid in bacillary infections; of neoarsphenamine in lesions due to cocci, and of sulfanilamide in the presence of gram negative bacilli, *B. proteus,* streptococci (except *S. fecalis*), and staphylococci. While this last agent is still undergoing intensive study, there seems to be definite evidence of its value. The need for eliminating stasis cannot be forgotten in dealing with resistant infections.

On the last day, **Dr. O. A. Nelson,** of Seattle, called attention to the frequency with which chronic infection of the uterine cervix produces urinary frequency. Granular urethritis is usually found. In his 87 cases, cauterization of the cervix done in 43 failed to relieve the symptoms. In 36 cases, the Sturmdorf operation was done, with 8 failures.

This method ought to be borne in mind in those patients whose granular urethritis is not relieved by ordinary methods (dilation, topical applications, heat).

Dr. William M. Coppridge, of Durham, N. C., reported a case of spontaneous subcapsular hematoma. This rare condition was recognized by Willis in 1700. In the acute form there is pain in the renal area with the formation of a mass and evidence of internal hemorrhage. In the chronic form a painless mass is associated with secondary anemia. Nephrectomy is often required. Microscopic examination oftenest shows chronic glomerulonephritis.

Dr. Richard Chute and **Dr. Sylvester Kelley,** of Boston, called attention to the frequency with which lesions of the kidney and ureter reproduce the symptoms and findings of gastrointestinal disorders, particularly appendicitis; and they recommended careful urographic study whenever there is anything atypical about the symptoms or findings in what appears to be gastrointestinal disease.

Dr. Norris J. Heckel and **Willard O. Thompson**, of Chicago, reported their very interesting experiences with the use of anterior pituitary-like substance for undescended testis in 28 patients. Descent under treatment occurred in 25 per cent, all of whom were under thirteen years of age. Failures were found at operation to be due to abnormalities of the structures surrounding the testis.

Most interesting was their observation of enlargement of the genitals which was just noticeable in 4 and very evident in 11 patients. The enlargement appeared only after nine or more weeks of treatment with a dosage varying from 100 to 1,000 units three to six times weekly.

The authors suggest that this regime may prove useful as a preliminary to orchidopexy, to supplement unsuccessful operation, enlarge the penis before operation for hypospadias, and in Fröhlich's syndrome (the genitals increased in size in 9 of their 12 cases of this type).

Dr. Charles B. Huggins, of Chicago, treated spermatocele in 9 elderly men with x-ray with success. He cautioned against its use in young men, and shielded the other testis with lead during treatment.

Dr. G. R. Livermore, of Memphis, treats hydrocele by aspiration with a trocar through which linen tape soaked in sodium morrhuate is then introduced; 15 out of 16 cases were cured.

Dr. Grayson Carroll, **Dr. Bransford Lewis**, and **Dr. Louis Kappel**, St. Louis, found by in vitro experiments that mandelic can kill *B. proteus* and *Eschericia coli*, while aerogenes and pyocyaneus survive even the "optimum" pH of five and concentration of 1 per cent. They claimed that 72 per cent of all cases treated with mandelic acid were cured.

Dr. Russell Herrold, of Chicago, reported that sulfanilamide (prontylin), although it failed in vitro to kill *Eschericia coli* in a concentration of 1 in 120, nevertheless cured 75 per cent of infections in the bladder and kidneys. It was ineffective in the presence of urinary stasis and calculi. Moderate reactions were frequent but not serious. He regards it as a "helpful adjunct" in chronic pyogenic prostatitis. It is his initial impression that sulfanilamide is a more effective urinary antiseptic than is mandelic acid.

The symposium on pyogenic prostatitis was not productive of any new data.

Dr. J. Sidney Ritter and **Dr. Charles Lippow**, of New York, made histologic studies of the prostates of guinea pigs and humans after the infection of electrargol as advocated by McCarthy. They, unlike O'Connor, saw no evidence of untoward effects.

Dr. Robert E. Cumming, of Detroit, is skeptical of the value of massage, heat, and intraprostatic injections. He recommends neoarsphenamine and antistaphylococcic serum for infections with staphylococci, and sulfanilamide for streptococci. When these measures were preceded by the eradication of other foci of infection, 75 per cent of his patients were relieved.

Dr. Henry A. Kreutzmann, of San Francisco, made cultures from the lumen of the vas deferens and found them positive in 40 per cent of patients with chronic prostatitis, cystitis, and in those recently subjected to urethral instrumentation.

These findings corroborate the popular idea that the epididymis becomes infected from the prostate by way of the lumen of the vas deferens.

Dr. Owsley Grant, of Louisville, discussed the treatment of "resistant" chronic prostatitis by the intraprostatic injection of 1 per cent mercurochrome, a measure with which he is very favorably impressed.

REVIEW OF THE FORTY-SECOND ANNUAL SESSION OF THE AMERICAN ACADEMY OF OTOLARYNGOLOGY

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THERE were many interesting and instructive papers presented at this meeting. Of the many papers there were some of interest to the surgeon, both in regard to surgery and in the cooperation the surgeon and otolaryngologist have established for the benefit of the patient.

A Brief Summary of the Unsolved Problems in Otolaryngology by **Dr. H. P. Mosher,** Boston, Mass. There are many questions unanswered, still, many interesting observations, which help to throw light upon some of these, but in the main there is much left to do. One problem has advanced and is partially overcome; namely, osteomyelitis of the frontal bone. Most of such cases result from an acute infection or from an exacerbation of a chronic frontal sinusitis. Much work has been done by Dr. Mosher and his staff advocating radical surgery with removal of the diseased bone including one-half to one inch of the surrounding healthy bone. With the established diagnosis and the knowledge that the infection has traveled one inch beyond the area of edema of the scalp, this radical procedure has met with good results and the mortality rate has greatly decreased. Not only should the anterior wall of the frontal be removed but the posterior wall as well, leaving the floor. A minimum of nasal surgery is done to the ethmoids at this time, only enough for established drainage through the anterior group of cells.

A Review of Nine Cases of Malignant Disease of the Nasal Accessory Sinuses was presented by **Dr. Frank R. Spencer,** Boulder, Colo., and **Dr. W. C. Black,** Denver, Colo. Nasal blockage and bleeding are the outstanding points in diagnosis, bleeding being the earliest sign. When nasal obstruction, swelling, pain, and foul discharge are complained of, the case has usually progressed beyond the stage of a hoped-for cure and only palliative surgery and deep therapy help to prolong the condition and relieve the symptoms, rather than removal or destruction of the growth. Of the malignancies epidermoid carcinoma is the most prevalent; and where it originates in the antrum or ethmoid the metastasis is by direct extension rather than by lymphatic or blood stream invasion. Here surgery has offered good results, but in the main, results are only partial; x-ray, too, has arrested the growth for a time. Where x-ray and surgery have been combined, the results have been favorable with the reports of many cures. The authors mention x-ray first because the vessels and lymphatics are sclerosed and sealed; there is less bleeding at the time of operation and less danger of metastasis through the lymphatics due to operative manipulation of the growth. Also, following operation, another period of x-ray treatment may be given within a reasonable time without danger to the patient and with much better effects than one massive postoperative dose.

Plastic Repair of Deformities About the Lower Part of the Nose Resulting From Loss of Tissue was presented by **Dr. V. H. Kazanjian,** Boston, Mass. He divides the amount of loss into four groups: first, the loss of only the skin of the nose; second, the loss of the tip; third, the loss of the tip and alae; and fourth, the loss of the mucous membrane of the nose. The causes are traumatic injury, carcinoma, angioma, tuberculosis, and syphilis. Before repair is attempted, all

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pathologic and scar tissues are removed. Following this a graft or pedicle is used. If a graft is used, full thickness is advocated. If a flap is the choice, it may be taken from any one of a number of places; arm, chest, or back. Frequently a graft from the forehead is brought down to the tip of the nose, but this leaves an area of denuded surface which has to be skin-grafted later and there is a certain amount of scarring. A graft from just below and posterior to the ear is the one of choice. Here there is no hair and the skin of this area has the same texture, thickness, and color as that of the nose. There is a negligible scar and it is in a location not readily noticed.

Treatment of Chronic Laryngitis Simplex by **Dr. Louis H. Clerf,** Philadelphia, Pa., was an interesting paper to those interested in surgery of chest conditions. Chronic laryngitis per se is rare. Ruling out the nose, throat, and nasopharynx, the next most likely focus is the chest. Bronchiectasis, tuberculosis, carcinoma, and benign tumors from their positions act as constant irritants to the larynx through coughing and expectoration. Provided the nose, throat, and pharynx are ruled out, every case of chronic laryngitis should have an x-ray of the chest; it is surprising how many chest conditions are picked up in this manner. Carcinomas at an early stage, after bronchoscopic biopsy, have been removed by the surgeon. Chronic laryngitis has led to the finding of severe bronchiectasis; and following lobectomy or pneumonectomy, the patient has gone back to a normal life. Whereas the bronchiectasis was the main factor, the laryngitis is only secondary.

The Panel Discussion of Septic Sinus Thrombophlebitis was led by **Dr. L. W. Dean,** St. Louis, Mo., **Dr. George Tobey,** Boston, Mass., **Dr. A. C. Furstenburg,** Ann Arbor, Mich., **Dr. Samuel Kopetzky,** New York, N. Y., **Dr. H. I. Lillie,** Rochester, Minn., **Dr. Allen Hartmann,** St. Louis, Mo., and **Dr. John MacKenzie Brown,** Los Angeles, Calif. This question has long been a topic of discussion throughout the various hospitals and medical centers of the country and an attempt has been made to arrive at an understanding between the different sections.

In the question on diagnosis there is no set rule, cases being reported without having had any external aural discharge so that like many other surgical conditions a careful history followed by a careful examination is our only lead. At the sudden onset of a lateral sinus involvement, there may be the characteristic chill followed by picket-fence temperature which fluctuates frequently within twenty-four hours. On the other hand the temperature may remain elevated with slight remissions, never reaching normal but varying little from time to time. Again, before the sinus is thrombosed, where there is a perisinus abscess, we may have remissions for twenty-four to forty-eight hours, followed by a slight rise due to absorption. Normal temperatures have been seen by all in the slow chronic type of infection where no involvement of the sinus is expected until the plate is uncovered. Blood counts help to rule out the lung and other fields; the higher the count the more likely one is to encounter other complications. The blood culture may be negative early, since the infection is fed through the small vessels and only later becomes positive as the sinus itself becomes involved. Where it has become involved by direct extension, however, the blood culture is positive early and the prognosis more guarded.

From an anatomic standpoint, few anomalies are met on the operating table; most are seen on cadavers or in the general routine postmortems. The sinus may end blindly near the knee, the petrosals taking its place; again it may be very small with hypertrophy of the emissary; on the other hand a very large sinus may be present at the expense of opposite side. One case was reported where it pierced the skull and appeared for a short time beneath the scalp before entering the skull again.

As regards treatment (whether to tie or not), a question still remains, left to the judgment of the operator. If not thrombosed with a septicemia but definitely infected, then the jugular should be tied. Where the infection has extended along the jugular vein, that vein should be tied and severed. With an infected thrombus involving the sinus, the sinus is opened; the clot is removed until there is free bleeding from both ends and is then packed off and obliterated; the question of tying the jugular is left to the judgment of the operator. Over a series of cases there is little to choose from in regard to tying this type as shown by the case reports, and the option is again left with the operator.

Because a sinus is thrombosed does not mean one has to rush in to any prolonged and tedious operation, requiring a good bit of skill and fraught with grave consequences and complications. The sinus is to be opened if the thrombus is infected; the jugular may or may not be tied, but the first regard is the care and condition of the patient so that when the operation is undertaken one starts with as good a surgical risk as is possible under the circumstances.

Book Reviews

Ross and Carless Manual of Surgery. By Cecil P. G. Wakeley and John B. Hunter. Edited by William T. Coughlin. Ed. American (15). Cloth. Pp. 1,536, with 900 illustrations. Baltimore, Md., 1937, William Wood & Company. $9.

The fifteenth American edition of this well-known English textbook, edited by W. T. Coughlin, purports to be a reliable textbook for students and general practitioners. Almost all subjects of practical surgical interest, except those of a gynecologic nature, are included within its 1,536 pages. The reviewer is somewhat disappointed, however, in failing to find many of the more recent refinements of surgical diagnosis and treatment with which the American surgeon is familiar. This failure to reflect adequately the recent progress in methods of diagnosis and treatment is the greatest criticism which can be made of the otherwise complete and admirable book. To cite specific examples: It is startling, to say the least, to find early incision recommended for cellulitis. The lack of emphasis accorded the roentgenologic examinations in the diagnosis of surgical conditions is typified by the absence of any mention of such examination in the section dealing with the diagnosis of intestinal obstruction. No mention is made of the injection treatment of inguinal hernia, nor of the conservative treatment of gastrointestinal stasis by suction applied to an inlying duodenal tube. The treatment of circulatory insufficiency by passive vascular exercises is omitted.

The book is written in a clear, concise style. The material is well organized and divided with many well-chosen subtitles. The illustrations are abundant and excellent.

Certain chapters are particularly good, especially those on "Injuries and Diseases of Arteries—Aneurism—Ligature of Arteries," "Injuries of Bones—Fractures," "Affections of the Brain and Its Membranes," "Diseases of the Breast," and "Amputations." The chapter entitled "The Use of Physical Agencies in Surgery" could be read with profit by anyone who has to deal with surgical conditions.

One wonders why the sections on gynecology have been omitted from the American edition when complete chapters dealing with surgical affections of the ear, nose, and throat have been included. An adequate index is appended.

Gastroscopy. By Rudolph Schindler. Cloth. Pp. 343, with 96 illustrations in color. Chicago, 1937, University of Chicago Press. $7.50.

This monograph is the first in the English language devoted to gastroscopy. The presentation of this subject by Dr. Schindler, an acknowledged authority on gastroscopy, should do much to popularize the use of this valuable addition to our armamentarium for gastroenterologic diagnosis. The author firmly establishes the fact that gastroscopy and roentgenologic methods when employed together are superior to any and all types of functional diagnosis, but one cannot safely be used with exclusion of the other.

The first portion of the book includes a comprehensive historical review of the development of gastroscopy and a study of the anatomy of the stomach from a gastroscopic view-point. Considerable space is devoted to details of technique which will prove invaluable to the beginner as well as to the more experienced gastroscopist.

The chapter on chronic gastroduodenal ulcer presents the various pictures of the development of an ulcer, its complications, and the healing of an ulcer. The subject of gastritis is fully discussed. Inasmuch as this is the most common gastroscopic finding in the average clinical material, this means of study should throw considerable light on the controversial subject.

A chapter on tumors stresses the early diagnosis of carcinoma and its differentiation from benign ulcer and indicates that gastroscopic examination should bring to surgery earlier those cases in which determination of malignancy is difficult by other means. In the later cases this examination in conjunction with x-ray should permit a more accurate selection of cases for surgery, thus reducing the number of exploratory operations in the inoperable cases.

An interesting series of observations on the postoperative stomachs of both satisfactory and unsatisfactory end-result cases is presented. Studies of the motility, postoperative gastritis, recurrent and gastrojejunal ulcer are very instructive.

The book is well illustrated throughout and the numerous colored pictures in the appended atlas are especially commendable.

Because of the author's original work and extensive experience, this authoritative presentation of the endoscopic study of gastric pathology will be a valuable addition to any medical library. Its straight-forward style should evoke considerable discussion on the pathology of the stomach and stimulate interest in gastroscopic examinations.

Inhalation Anesthesia—A Fundamental Guide. By Arthur E. Guedel. Cloth. Pp. 172. New York, 1937, The Macmillan Company. $2.50.

The foreword by Ralph M. Waters, professor of anesthesia, University of Wisconsin, outlines the author's rich background of experience and teaching out of which the book grew, and states: "As a result, the author has evolved a technique of teaching which has been found exceptionally fruitful in the hands of others as well as himself in the instruction of undergraduate and graduate medical students. The methods of Guedel have long been used by many of us. Personally, I am very happy at the prospect of having available the contents of this small book as an aid in my attempts to convey to medical students and young physicians the principles and practice of inhalation anesthesia."

The book differs considerably from those on anesthesia previously published. It is confined entirely to inhalation anesthesia. It does not deal too minutely with theory, but it presents all necessary facts of physics and physiology to make the mechanisms, the signs, and the favorable and unfavorable reactions in anesthesia understandable. The technique of open ether administration is given as a pattern in quite complete detail, together with the attendant physical mechanisms of vaporization, concentration, vapor pressures, pulmonary absorption, distribution, blood and tissue concentration and retention. One is told just how to begin the induction with open drop ether and just how to vary the administration and technique in response to various reactions. The description is well given and helpful to the student. The description of semiclosed and closed methods with both liquids and gases is dismissed with the simple statements that the principles are the same except that the carbon dioxide is disposed of by allowing partial escape or partial or complete absorption by soda-lime, and that liberated nitrogen dilutes the gases and should be dispensed with by occasionally emptying the bag. However, the author is able to give good ether instruction without lessening the emphasis on principles and one wishes he had given the same concerning the closed methods. If it is helpful and desirable to give one, it should be helpful and desirable to give the other.

The author presents beautifully the stages and planes of anesthesia, the signs of anesthesia, the depths of anesthesia required for control of reflexes associated with the various structures of the body, and the classification of surgical procedures which can be accomplished under the various planes of anesthesia.

A chapter is devoted to the mechanism of the anesthetic requirements of the patient, his resistance and susceptibility to anesthesia, based upon his metabolic rate and the factors, such as endocrine imbalance, fever, pain, emotional excitement, which chronically or acutely influence the rate. The effects and usefulness of the various drugs used in preparation for anesthesia are discussed, especially as to their influence upon the metabolic starting point of anesthesia. The potencies of the various inhalation anesthetics are estimated and the planes of anesthesia which may be obtained by them in relation to the starting point.

An especially admirable and outstanding feature of the book is Part II, more than half of the pages, devoted to anesthetic accidents. The author discusses accidents due to changes in blood pressure—such as, acute cardiac dilatation, rupture of a blood vessel, cerebral anemia, cardiac anemia; ventricular fibrillation; nitrous oxide in obstetrics; central respiratory failure caused by overdose of preanesthetic drug or anesthetic, increased intracranial pressure or cerebral anemia; accidents due to peripheral respiratory interference, breath-holding, pharyngeal spasm, laryngeal spasm, tongue swallowing, aspiration of débris, vomitus, mucus, pus from pharyngeal or peritonsillar abscesses, bronchiectatic débris in lung surgery, blood from surgery of the nose, throat, and mouth, teeth or particles from broken teeth, sponges, instruments; miscellaneous accidents, massive atelectasis, tracheal collapse, convulsions under anesthesia, status lymphaticus, embolism, idiopathic paroxysmal tachycardia, liquid ether into the lungs, the vapor of heated ether, injuries to the eyes, postoperative hyperthermia, cerebral asphyxia; cyanosis, the causes, degrees, and significance of cyanosis and factors which influence its observation. An abundance of illustrative cases give a very practical and instructive aspect to this part of the book.

The final chapter treats with anesthetic explosions and contains information as complete and simply stated as is available concerning the causes and methods of preventing these accidents. The book is very readable and will be a great help to any one who teaches, practices, or studies inhalation anesthesia.

Textbook of Surgical Nursing. By Henry S. Brookes, Jr. Pp. 636, with 233 illustrations. St. Louis, 1937, The C. V. Mosby Company. $3.50.

This is an entirely new book, compiled and written principally by Dr. Henry S. Brookes, Jr., with contributions by Drs. H. S. Crossen, T. P. Brookes, E. A. Graham, and Ernest Sachs.

In the first chapter the author discusses the attitude and approach on the part of the nurse which are most likely to establish ideal relationships between her, her patient, and other persons with whom she comes in contact while doing surgical nursing.

The remaining portion of the book follows the form usually used in general surgical texts, with special chapters on inflammation and surgical infections, burns and skin grafts, ulcers, gangrene and necrosis, wounds, tumors, dressings, bandages and binders, dressing room technique, first aid, preoperative and postoperative care, and anesthesia. Operating room and other division duties of a surgical nurse are presented in detail, and the routine and special procedures peculiar to nursing care of surgical cases are discussed. There are adequate discussions of the etiology, the

surgical anatomy, the pathologic physiology, the histopathology, and the operative procedures involved in the various surgical diseases.

The methods of procedure discussed are those presently in vogue at Barnes Hospital, St. Louis, Mo., and, as the author observes, some are neither applicable nor in accord with established practices elsewhere. The general principles, however, and in most instances the detailed recommendations made in this book are generally accepted as excellent.

Selected medico-legal points especially pertinent to surgical nursing are presented in a short chapter. A glossary, including many of the terms used in the discussion or the directing of surgical cases, might be referred to advantageously, especially by nurses just beginning their training in a surgical division. The illustrations are good and depict modern methods and equipment.

A Manual of Radiological Diagnosis. By Ivan C. C. Tchaperoff. Pp. 256, with 286 illustrations. Baltimore, 1937, William Wood & Company. $6.

This profusely illustrated outline of roentgen diagnosis should find ready acceptance by undergraduate students and practitioners of medicine. It presents the essentials of radiologic diagnosis in brief form. The best section in the book deals with the bones and joints and occupies about half of the volume. The chest, gastrointestinal tract, gallbladder, urinary tract, female generative organs, including the radiologic diagnosis of pregnancy and tumor of the spinal cord and ventriculography, all receive consideration but in somewhat briefer form. The book is modern and should prove to be a valuable manual for quick reference.

Erratum

On page 528 of the October issue, in the article by Scudder and Zwemer entitled "The Effect of Complete Intestinal Fistula on Blood Potassium," in the sixth paragraph reference should be made to Morton's hypothesis rather than to Norton's.